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THE "DISCHARGING LESION" IN NEUROLOGY.[1]

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I. INTRODUCTION.

THE hypothesis of the "discharging lesion" propounded by Hughlings Jackson to account for convulsive phenomena has received very little treatment, except in his own writings, since it was first put forward. It is, however, capable of amplification and extension and seems to me to have a much wider application in the theory of clinical neurology than has hitherto been accorded to it. There are motor phenomena other than convulsions in which we have before our eyes objective evidence of less violent abnormal discharges—as, for instance, the twitching of peripheral facial spasm, the muscular fibrillation of amyotrophic lateral sclerosis, the trismus of tetanus—and there are sensory phenomena which it seems best to interpret as examples of the same effect at work in sensory systems and especially in the pain system: there are also other symptoms which I feel are best explained as due to similar irregular discharges exciting reflex activities. As a clinician I will base my deductions on clinical observation, but it will unify what I have to say and provide a better appreciation of the underlying problem, if I begin with a consideration of Hughlings Jackson's theory.

II. HUGHLINGS JACKSON'S THEORY.

On the subject of the "discharging lesion" in its relationship to convulsions—and especially local convulsions—Hughlings Jackson wrote

[1]Presidential Address to the Neurological Section of the Royal Society of Medicine, October 4, 1945.

voluminously and we may return to his writings (Jackson, 1931) for statements of the theory in its classical form, and we may observe at the same time the development of it in his own mind. He began his best-known paper, "A Study of Convulsions," as follows (p. 8):—

> "A convulsion is but a symptom, and implies only that there is an occasional, an excessive, and a disorderly discharge of nerve tissue on muscles. This discharge occurs in all degrees: it occurs with all sorts of conditions of ill-health, at all ages, and under innumerable circumstances."[1]

Later in the same paper when he refers to *post-mortem* findings in cases of local convulsions, he gives an expanded statement of the general theory, which is perhaps the best-known passage in the whole of his writings (p. 25):—

> "It is the function of nerve tissue in health to 'store up' force and to expend it in an orderly manner at the provocation of special excitations. The discharge of disease differs from the expenditure of nerve force in health, in quantity and in that it is provoked by a more general excitation. And even in those cases in which we do find a lump in the brain—we do not discover the *very* change on which the discharge depends. The lump does not discharge, but some ('softened') part of the brain near it—which part cannot be destroyed, or it would not discharge at all, but which part must be diseased or it would not discharge as much, nor in so disorderly a manner, nor on slight provocation."

Six years later, trying to be more precise, he tells us in a footnote (p. 211) that such expressions as "store up force" are used conventionally and that he had no theory as to "nerve force" or force of any kind. Since then mechanics has given the word "force" a somewhat different meaning, but it is obvious that he used it in the sense of the modern word "energy," and so read, this statement made seventy-five years ago, is very close to to-day's ideas of the truth.

In a later paper he wrote (p. 94):—

> ". . . we say there is in a case of epilepsy grey matter which is so abnormally nourished that it occasionally reaches very high tension and very unstable equilibrium and therefore occasionally explodes."

And again (p. 97):—

> "I believe then, that the highly unstable nervous matter of disease (in a 'discharging lesion') differs in composition, but not in constitu-

[1]The page references are to Vol. I of the "Selected Writings" of Hughlings Jackson, edited by James Taylor.

> tion, from the comparatively stable grey matter of health. The alteration in composition is, of course, such that the nervous substance formed is more explosive. We must suppose that there is some order in this Substitution—Nutrition, and we must infer that it is in the direction of explosiveness, or instability."

Finally, twenty years later, in his Lumleian Lectures he phrased it thus (p. 413):—

> "I have always assumed, and shall continue to suppose, that convulsion results from excessive discharge of nerve cells, meaning, of course, liberation of energy during rapid decomposition (katabolism) of some matter in, or of part of, those cells. I shall frequently speak of cells concerned with excessive (primary) discharges as constituting a 'discharging lesion'. . ."

Here he has gone a step further and has, moreover, given us his notion of how the energy is "stored up" in the cell, viz.: as "some matter" which during rapid decomposition liberates energy. Perhaps we may call it simply an "energy substance"—glucose as it might be or gunpowder if you like!—some chemical substance which can break down rapidly and is a convenient source of energy.

It is obvious that the excessive discharges he had in mind were in nature the same as the physiological discharge of the nerve cell. Although I do not recall that it is explicitly stated, Hughlings Jackson evidently supposed that the nerve cell made use of the energy of its "energy substance," and transformed it to provide the energy of its own specific impulses—which we know now to be very largely electrical. It is thus inherent in the theory that it is a function of the nerve cell—as perhaps it is of every cell—to transmute one form of energy into another.

There then is Hughlings Jackson's theory. It has two essential portions, which, using modern terms and interpreting his statements generously, we may express as follows: (1) The nerve cell contains, or has accessible to it, a store of energy in the form of a chemical "energy substance"—which we have likened to glucose—and, "at the provocation of special excitations," i.e. in response to physiological stimuli, it expends energy in an orderly manner in the form of its own specific impulses. (2) Under some abnormal conditions, stored or available energy may be released, without any stimulus of a physiological character (he speaks of "primary" discharges) and under such conditions the discharges are excessive and disorderly. I would have you notice again that they are still of the nature of nervous impulses. The "energy substance," we may suppose, breaks down under abnormal conditions of environment, and

in doing so causes the cell to perform its physiological action, albeit in an excessive and disorderly manner. Using a phrase with a wider connotation we may say that the energy is "discharged through its physiological channel."

Before we leave Hughlings Jackson, it may interest you to notice how he used the term "discharging lesion." It was a term rather wittily borrowed from the surgical wards. It does not occur in "A Study of Convulsions" which appeared in print in 1870. It is used for the first time, as far as I have discovered, in a paper on the investigation of epilepsies which appeared in 1873 (p. 94), and from then on it occurs frequently, the "discharging lesion" being often contrasted with the "destroying lesion." To the end of his days Jackson wrote it in inverted commas, at any rate, when first used in an article, though by 1890 he could bring himself to drop the inverted commas after the term had, so to speak, been properly introduced. But as early as 1873 we find Ferrier who was more forthright writing without any inverted commas or qualification of "the destroying and discharging lesions of disease which his (i.e. Hughlings Jackson's) writings have defined and differentiated."

III. Its Relation to Modern Physiological Knowledge.

Now let us see how this theory stands to-day. Is it able to keep afloat in the spate of scientific discovery of all kinds that has flowed from physiological laboratories in the intervening years? It would obviously not be worth my while to pursue it further if it conflicted with well-substantiated physiological knowledge. In fact the opposite is the case. Far from being in conflict modern knowledge helps to support and develop the theory. To-day's view attributes to all cells some store of energy and the power to transform and adapt it to their own several purposes. To quote from Sherrington (1940): "Essential for any conception of the cell is that it is no static system. It is a material system and that to-day is to say an energy-system. Our conceptions of it fail if not dynamic . . . yet each and every step is understandable chemistry. The cell has proved to be a perfect swarm of catalysts, or of trains of catalysts, each a link in a serial suite of chemical action" ("Man on his Nature," p. 78). Of the nerve cell in particular we realize from general principles that it has energy immediately available, whereby it relays whatever messages it receives, augmenting them perhaps tenfold, perhaps a hundredfold. To quote another physiologist: "It seems beyond doubt that energy is evolved in the central nervous system as a result of metabolic changes, and that energy may be added to impulses passing through the central nervous

system, which therefore acts as a relay of force" (Hartridge, 1930). Unfortunately for our theory physiological work on fundamental processes in the nervous system in recent years has proceeded along lines of physics rather than chemistry, but some little is known of the chemical accompaniments and although the amounts of energy involved in the transmission of nervous impulses turn out to be very small, that is not to say that they are any the less important (cf. Hill, 1932).

The cell in regard to which our knowledge is most advanced in this respect is the muscle cell, one which has many points of similarity with the nerve cell.

In my early days in neurology, I knew nothing of Hughlings Jackson's teaching except the merest scraps. I had great difficulty in formulating for myself any "rationale" for convulsive phenomena, but I eventually came to an almost identical hypothesis by way of the physiology of muscle, my mind doubtless prepared by what I had heard of Jackson's ideas. The muscle cell possesses essentially just those properties that Jackson postulated for the nerve cell. Muscle is ready to do work at any moment and so it must possess a store of energy. It happened that in 1925 I attended a course of lectures given by Professor A. V. Hill on the physiology of muscle. Hill at that time had just finished that brilliant series of investigations into the metabolism and dynamics of muscle, an account of which was subsequently summarized in his Herter lectures (Hill, 1926), and in the lectures which I attended he gave a series of demonstrations relating to the dynamics of muscle. It was shown: (1) that the muscle cell contained a store of energy, which it could draw on at any moment for the performance of work; (2) that it possessed this store by reason of a content of an "energy substance" (allied to glycogen) the katabolism of which released chemical energy which the muscle was able to transform into the physical energy of work and heat; (3) that a physiological stimulus led to the breakdown of a controlled amount of thin energy substance, the energy thus rendered available being expended in doing work—the physiological action of the muscle cell—and in the liberation of heat; (4) when the muscle rested its energy substance was fairly quickly restored and even during activity restoration was going on. But now here is the fact that is important for our present purpose, and for the second portion of Hughlings Jackson's theory: quite apart from any stimulus of a physiological kind there were many circumstances which caused the muscle to go into spasm—into rigor—heat, absence of circulation, the action of organic salts, the action of caffein and so on—but in every instance there was an expenditure of the energy substance, corresponding in amount to

the work done and the heat liberated. ". . . no means of separating the metabolic changes and the external mechanical changes has ever been found" (Hill, 1926). Even with the extraordinary peculiarities of the veratrin contraction this rule obtained.

A piece of muscle could be regarded as a group of muscle cells, and the point on which my mind seized was that under certain environmental conditions which led to a disturbance of its metabolism the energy substance of the cells broke down "spontaneously" (i.e. in the absence of physiological stimulus), with consequent discharge of energy, and that its breakdown was associated with the performance, albeit in a perverted manner, of the physiological action of the cell. Under such circumstances, in contrast to physiological activity, both metabolism and action were uncontrolled.

The features were thus those of a "discharging lesion." The spasm of the muscle, with its expenditure of physical energy, was the outward and visible sign of the breakdown of the energy substance within the cells, or in other words of the expenditure of their store of energy.

Is not this almost exactly what Hughlings Jackson postulated for the nerve cell? "It is the function of nerve tissue in health to 'store up' force and to expend it in an orderly manner at the provocation of special excitations." ". . . liberation of energy during rapid decomposition (katabolism) of some matter in . . . those cells."

As I have said I knew little of all this, but it was not a big step after Hill's lectures to ask myself whether the nerve cell also had a store of energy and an energy substance. It was known that brain tissue made use of a considerable amount of glucose, and I began tentatively to interpret symptoms on the supposition that the nerve cell contained an energy substance. I soon found that I had to suppose that nerve fibres and tracts must also possess such a substance, because under pathological conditions impulses evidently originated in them. Some years afterwards I discovered that Hughlings Jackson had made the same supposition (p. 211)—as indeed anyone must who thinks along these lines.

Since that time it has been shown that the nerve fibre is comparable as Adrian has expressed it not so much to a telephone wire (which merely conveys a current) as to a train of gunpowder—a system which in some degree expends itself in conveying the impulse. "The energy involved in the transmission of the impulse is certainly derived from the potential energy present in the resting fibre and not from the stimulus" (Adrian, 1928). "We may liken it (the impulse) to an explosive wave started by the trigger action of the stimulation" (*ibid.*).

Shortly before the recent war A. V. Hill, by thermo-dynamic methods similar to those which he had used so successfully on his work on muscle, actually estimated the energy expended by a length of nerve in transmitting an impulse.

The first direct demonstration of "spontaneous" discharge in nerve tissue under pathological conditions came from Adrian's laboratory in the discovery that in an injured nerve a stream of impulses originated at the site of injury (injury current) and under favourable conditions might continue for a very long time (Adrian, 1935), and this must be the first discharging lesion from which the immediate discharges have been recorded. The laboratory thus demonstrated in its way something which the statements of our patients, and even our own bodies, had forced on our belief long ago. There has not to my knowledge been any such direct demonstration of "spontaneous" discharges from nerve cell bodies under pathological conditions. The electro-encephalograph gives frequent evidence of excessive and disorderly discharges from portions of cerebral cortex in abnormal states.

The present state of the theory, therefore, as regards support from the laboratory, is (1) that it seems established that the *nerve fibre* contains an "energy substance," and the energy of its metabolism has even been estimated. "Spontaneous" discharge from injured nerve has been demonstrated. (2) As regards the cell body, it evidently contains a store of energy in some form and under pathological conditions disorderly discharges occur from it. The muscle cell with which it has many similarities contains a store of energy in the form of an "energy-substance" and under certain abnormal conditions this energy substance may break down, with consequent expenditure of some or all of the energy content of the cell; this expenditure of the energy substance is accompanied by a perverted physiological activity of the cell.

IV. Some Features of the Objective Manifestations of Discharging Lesions.

My principal concern, however, is not with the theoretical basis of discharging lesions but with their clinical characteristics, for in the present state of knowledge it is on these latter that we must base any further applications of the theory. There are many symptoms which present us with objective evidence of irregular, disorderly and, as far as we can see, spontaneous discharges. We must review the features of these before applying the theory to subjective phenomena, and in doing so we shall

find ourselves compelled to make certain modifications in the Jacksonian teaching.

Varieties of Discharge.

Hughlings Jackson wrote on convulsive phenomena at great length and I wish to make only brief further reference to them. He supposed that a local convulsion resulted from some portion of grey matter becoming "overcharged," e.g. "it occasionally reaches very high tension" (p. 177) and discharges. For him the violent paroxysm of the convulsion resulted from a massive or explosive type of discharge from overcharged cells. To-day it seems unnecessary to make the assumption that the cells involved are in any way excessively charged: it is sufficient to suppose that as a result of changes in the cells environment their energy substance breaks down, more or less rapidly. Nevertheless the discharge that gives rise to a violent convulsive attack may be described as massive or explosive.

Other discharges, however, are less violent. Some years ago I had under my care a Lascar seaman suffering from cerebral syphilis in whom local fits affecting the right side of the face, tongue and right hand occurred many times daily from the time of his admission to hospital, and after a number of days twitching of the right side of the face became continuous and it persisted without cessation for a fortnight. During this period it was never violent and the discharge giving rise to it would be better described by the word "spluttering" than by "explosive." Other instances of "spluttering" discharges are those which give rise to peripheral facial spasm and those which cause the muscular fibrillation of amyotrophic lateral sclerosis.

In a third group, phenomena resulting from "spontaneous" discharge are of a continuous character. Of these the trismus of tetanus is perhaps the best example. Others are the carpo-pedal spasms of tetany (assuming that they are at least partly neurogenic) and a slight continuous spasm in one side of the face that is seen in some cases of eighth nerve tumour.

There is a fourth group of objective phenomena in which the activity is below the threshold of spontaneous discharge, but the motor responses to voluntary, emotional and other stimuli are excessive, e.g. certain spasms in tetany and in strychnine poisoning. This condition may be referred to as a "subliminal discharging state."

The Steady State.

From Jackson's supposition that some portion of nervous tissue became overcharged, it followed that discharge must be paroxysmal and occasional, and for him this was a fundamental character of the symptoms resulting

from a discharging lesion. His pupils too have mostly taken the view that phenomena resulting from spontaneous discharge (or from "irritation") were essentially transient. While it is true that the convulsive phenomena resulting from the more violent (explosive) discharges are transient, the same is by no means true of the symptoms which we have attributed to the "spluttering" and continuous discharges. Trismus, which we must attribute to an excessive and continuous nervous discharge, may persist for weeks. In the case of peripheral facial spasm each muscular twitch or flicker may be regarded as a paroxysm, but in a bad case the affected side of the face is never at rest and we must suppose that the lesion responsible, regarded as a whole, is in a continuous state of discharge and this persists for the rest of the patient's life. We are thus compelled to suppose that in certain discharging lesions expenditure of the "energy substance" may be counterbalanced by its restoration. This "steady state" is a well-known phenomenon in muscle, and, in fact, must occur under physiological conditions in all cells that are capable of prolonged activity. Provided then that the discharge is not of a violent character, there is no reason why symptoms due to a "spontaneous" discharge (from a discharging lesion) may not be prolonged or permanent.

Sites of Discharge.

The most violent discharges evidently take place at the cortical level. Hughlings Jackson deducted this by the clinico-pathological study of cases of localized cerebral lesions and much pathological and physiological evidence since then has confirmed it and in recent years further strong confirmation has been provided by the electro-encephalograph.

The less violent, spluttering and continuous discharges may evidently occur from different levels. In the case of the Lascar seaman to which I have referred and in many similar instances (e.g. epilepsia partialis continuans) the spluttering activity seems to be cortical. In the case of idiopathic peripheral facial spasm it may be in the facial nerve nucleus or in the nerve itself. In the case of the variety that follows Bell's palsy we know from the work of Balance and Duel (Balance, 1934) that there is a lesion of the nerve at the lower end of the facial canal and it seems most likely that the discharge which excites the facial twitching arises at this site, but it may be that it arises in cells of the facial nucleus as a consequence of secondary changes in them. It may be recalled, however, that Adrian's injury current is a phenomenon of peripheral nerve. As regards the spluttering activity responsible for the fibrillation of amyotrophic lateral sclerosis it seems most likely that it arises in the degenerating

anterior horn cells in the spinal cord, but other sites may be suggested.

In the case of the continuous spasm of trismus such indications as we have suggest that it depends on discharges arising at the pontine level, because it is bilateral and the facial musculature, innervated also from the pons, may be similarly affected. The nuclei would seem to be the most likely site of this "spontaneous" activity. We have little indication of the site of origin of the discharges responsible for the continuous spasms of tetany.

This is obviously a matter on which much more might be said and further evidence could be adduced from certain sensory phenomena, but without going into detail it is apparent that discharges may arise in structures at different physiological levels from the motor cortex to the peripheral nerve fibre.

Conditions Associated with "Spontaneous" Discharges.

Although it is seventy-five years since Jackson's "Study of Convulsions" appeared, we do not yet know the immediate exciting cause of the abnormal discharges in a case of Jacksonian (or any) epilepsy, nor have we yet identified even one special kind of pathological process that invariably gives rise to discharging lesions. Some oxygen deficiency is present in a number of discharging conditions and it is known that under conditions of physiological observation anoxia causes a state of hyperexcitability and discharge in many nervous structures, but it is not constantly present in the abnormal conditions we are concerned with and we must beware of drawing any further conclusion than the evidence warrants.

We may, however, note in passing some further circumstances connected with certain of these phenomena which provide us with objective evidence of abnormal discharges. I have already referred more than once to the twitching of one side of the face which not infrequently follows Bell's palsy and which may become severe, "paroxysmally continuous" and persistent. There are good grounds for attributing this twitching to discharges arising in the lower motor neurone, but whether primarily from the cells in the facial nucleus or from the fibres of the damaged nerve it is impossible to say. Whichever view is taken, it must be noticed that the twitchings do not begin until the process of recovery has made some considerable advance. Recovery, however, has not become complete, nor does it become complete (as evidenced by return of full voluntary movement) in those cases in which twitching persists. Whatever their cause and site of origin the discharges of which the twitchings are the

evidence take place in an imperfectly recovered nerve (i.e. its cells and/or fibres). Our theory would say that for some reason (for instance impairment of the circulation as a result of scarring) the energy substance in nerve fibres or cell bodies is not in stable equilibrium with its chemical environment: either the energy substance is not completely synthesized to its stable form or it is not adequately buffered, and this abnormal state persists.

The only example of the spluttering type of discharge of which I wish to make further mention is fibrillation (or fasciculation) of muscle as it occurs in amyotrophic lateral sclerosis. In this disease, as the anterior horn cells degenerate, irregular twitchings of fibres occur in the corresponding muscles. It is true that the muscles are degenerating, and so some of the disturbance may be essentially muscular, but the primary degeneration is in the cells of the spinal cord, and the size of the contractions suggests that they are due to impulses from the spinal cells, and, in fact, Denny-Brown and Pennybacker (1938) showed that they were associated with action currents in the motor nerve. We must, therefore, conclude that in this disease the degenerating anterior horn cells frequently emit little spluttering discharges—without, as far as we know, any physiological stimulus. (Even if discharges arise in the degenerating pyramidal fibres, the rest of the argument is the same.) It seems then that in this disease, cells, not in a state of imperfect recovery as in the last example, but in the process of a slow degeneration, remain liable to emit frequent small discharges without physiological stimulus—liable, as it were, to little spluttering explosions of their energy substance, i.e. that their energy substance is, over a long period, in a state of instability.

There are not many good clinical examples of discharge of the continuous variety from motor nerve cells. Tetanus provides one of them and the spasms seem *prima facie* to be due to an effect of the tetanus toxin on the trigeminal motor nuclei and the facial nuclei. When the spasm is prolonged breakdown and renewal of the energy substance are probably going on simultaneously in the cells of the affected nuclei, but in some cases the face becomes permanently paralysed, showing that the cells of the facial nucleus have been overwhelmed by the toxin and are then either incapable of rebuilding their energy substance or else of responding to it. A prolonged discharge has gone on in the course of a toxic process, and that toxic process may eventually lead to the death of the cell.

V. Applications of the Theory to Sensory Symptoms.

I have confined the discussion hitherto to discharges in motor struc-

tures, because in them they produce phenomena capable of objective observation. We have obtained evidence that under abnormal conditions "spontaneous" discharges may occur, that they may be of different degrees or varieties, that they may be paroxysmal or continuous, transient or persistent, that they may arise at different physiological levels from the cortex to the peripheral nerve, and that they may occur in a structure in a state of imperfect recovery or of slow degeneration, or under the action of a toxic process which may or may not in the end be lethal to the cell.

Many sensory phenomena which we experience ourselves, or more often which our patients describe to us, provide evidence of similar discharges occurring in sensory structures. Fits in the various sensory systems were described by Hughlings Jackson and attributed to paroxysmal discharges occurring spontaneously in the several areas of the cerebral cortex which subserve the primary sensory functions and at a lower physiological level we have the sudden intense discharges of the trigeminal neuralgia and of tabetic pains both of which he also referred to.

Pains which may be interpreted as results of spontaneous "spluttering" discharge in sensory cells are not uncommon as, for instance, the less severe pains that occur in trigeminal neuralgia and in tabes when the patients say the pains are "about." In the case of trigeminal neuralgia we know nothing of the pathology but in tabes we are dealing with a disease causing degeneration of afferent elements. The discharge may arise in a cell body of the posterior root ganglion or in a pain fibre of the peripheral nerve or posterior spinal root or in cell bodies within the spinal cord. Many of these elements subserving pain appreciation ultimately die, causing the tabetic hypalgesia. I conceive that the cell of the posterior root ganglion in a case of tabes when the patient is having frequent, though not very severe, pain, is in much the same state as the anterior horn cell in a case of amyotrophic lateral sclerosis when fibrillation is going on. Both are in a state of spluttering discharge and in both the liability to discharge is associated with a degenerative process.

The continuous type of "spontaneous" discharge is more common in sensory structures than in motor structures, but the spluttering type passes insensibly into the continuous. In other instances it seems that the energy substance is continuously in such a state of instability that, although it does not break down spontaneously, a physiological stimulus which would normally cause the breakdown of a "controlled" amount of it produces an excessive response (subliminal discharging state).

Phenomena indicative of "spontaneous" discharge at different physiological levels occur in different sensory systems (particularly those of the

special senses), but the symptoms which most call for consideration are those involving pain. Lesions at different levels from the peripheral nerve to the thalamus may be associated with pain—pain which is apparently spontaneous, which is usually continuous and is of a peculiarly insistent or compelling character—causalgia, post-herpetic neuralgia, spinal pains in syringomyelia, pains due to "irritation" of the spinothalamic tract (e.g. in cases of neurofibroma on an anterior spinal root), pain associated with lesions in the lateral nucleus of the thalamus. The response to a physiological stimulus in the area to which the pain is referred varies from nil in cases of complete analgesia to an intense tingling spreading pain, according to the degree of interruption of the conducting mechanism.

It is obvious that I cannot here consider these conditions in detail, and the application of the theory to most of them is clear, but it is necessary that I should say a little more about the thalamic syndrome.

The Thalamic Syndrome.

This syndrome has evoked a great deal of discussion since it was first described by Dejerine and Roussy in 1906. In 1911 Head and Holmes reinvestigated the problem of thalamic pain and put forward to account for it an hypothesis based on the theory of release of function which, in spite of increasing difficulties, has continued to hold the field. Riddoch, reconsidering the whole subject of Pain of Central Origin in his Lumleian Lectures of 1938 adhered to this hypothesis, though perhaps rather half-heartedly and with an obvious hankering after the theory of "irritation." Personally I have never been able to accept it, and I suggest that the condition in the thalamus is a discharging lesion.

The essential facts that have to be accounted for are not in dispute. The most constant phenomenon is that the patient reacts excessively to painful stimuli and to the more intense degrees of temperature, and in most cases he suffers in addition from spontaneous pain in a portion (or possibly all) of the affected half of the body. The threshold for pain is, however, not lowered, but on the contrary, is often raised, Head and Holmes finding such a condition in 9 out of 22 cases. In fact, in some instances there may be complete analgesia on a portion of the affected side, and yet the patient may complain of intense pain referred to the analgesic area. The painful sensations, however evoked, have a peculiar explosive, excessive, compelling quality. It is generally accepted that whether pain occurs only as an excessive reaction evoked by a stimulus or whether it also occurs apparently spontaneously is dependent on the degree of the abnormal state. In addition to the clinical features of the sensory

disturbance, it is a matter of general agreement that this peculiar sensory syndrome occurs almost exclusively as a consequence of lesions of a vascular nature and is rarely encountered in more than a rudimentary state with tumours.

To account for this syndrome Head and Holmes postulated that the sensory nuclei of the thalamus were normally controlled by higher centres in the cerebral cortex, and that when the cortical control was abolished by the interruption of "cortico-thalamic" fibres, the thalamus was "released" and acted excessively, just as spinal reflex centres act in an exaggerated manner when the pyramidal tract is interrupted.

Now, in the first place, it may be doubted whether the theory of release of lower centres is applicable at all to an ascending system. But, apart from that theoretical consideration, I find it difficult to make this hypothesis accord with the facts. In the cases referred to, the lesions are actually in the thalamus, and with suprathalamic lesions the syndrome does not develop, though it is obvious that lesions so situated and leaving the thalamus intact should permit the postulated release of the full function of that organ. Actually it is the centre which is supposed to be released which is damaged. Later, when it became apparent that lesions at a lower level might give rise to similar phenomena, the advocates of this theory were compelled to suppose either that the cortical inhibitory effect extended down to the next lower sensory neurone or, alternatively, that some other influences were operative. Head, at an earlier period, had, as you know, postulated two sensory systems extending right to the periphery, one of which was supposed to exercise an inhibitory effect on the other (Head, 1905). However, even with such hypotheses it was impossible to explain "spontaneous" pain and particularly spontaneous pain referred to areas which presented a high threshold for pain or were actually analgesic. It was argued that the pains are not really spontaneous. "Pains may be said to be 'spontaneous' only in so far as the stimuli which excite them are not obvious" (Riddoch, 1938), but even when that is said—and it is obviously difficult to accept for cases in which the threshold is high—the combination of continuous "spontaneous" pain and analgesia is difficult to explain and further suppositions have to be made to bolster up the theory. I am irresistibly reminded of Pasteur's paragraph on the subject of good and bad theories: "The characteristic of erroneous theories is the impossibility of ever foreseeing new facts; whenever such a fact is discovered those theories have to be grafted with further hypotheses in order to account for it" (Life of Pasteur, 1919, p. 243).

Now it seems to me much more satisfactory to postulate that imper-

fectly recovered cells in the thalamus constitute a "discharging lesion," that as in the imperfectly recovered fibres of the facial nerve, their energy substance remains in an unstable state. Where if not in a partly recovered vascular lesion might we postulate a state of (partial) anoxia? I suppose, furthermore, that the lesion attains a "steady state" in which the rate of break-down of the energy substance is compensated by its restoration, while, the lesion being actually in the thalamus, the raising of the threshold for pain and occasional analgesia are accounted for by damage to the terminal portions of the second sensory neurones which normally arborize with the cells so affected. This is the same as the explanation of anæsthesia dolorosa, in all instances, wherever its casual lesion may be.

I submit that this hypothesis is much more in accordance with the observed facts than one founded on release of a lower centre or withdrawal of an inhibitory influence. It has the advantages: (1) that it refers to a lesion actually in the thalamus which is what is found and not above the thalamus, where, as far as we know, the pain system does not extend; (2) it accounts for spontaneous pain as well as excessive response; (3) it accounts for the peculiar explosive quality of the response to painful stimuli; (4) it allows for a raised threshold to pain and even analgesia on areas of the body to which the pain is referred. Furthermore, it accounts, I think, for the fact that the syndrome occurs particularly with vascular lesions and develops in the recovery stage after the lesion. A similar theory is applicable to lesions in the pain system at lower levels, the abnormal discharges arising in fibres of the spinothalamic tracts or from associated cells in the cord, as, for instance, in syringomyelia. Holmes, in reviewing his experiences of spinal injuries in the war of 1914-18, seems to have adopted an explanation similar to this for the occurrence of pain in traumatic lesions of the spinal cord (Holmes, 1921).

Before we leave the thalamic syndrome it may be observed again that two different painful conditions are represented in it: (1) a pain which is spontaneous and though variable in intensity is continuous. (One of my patients, who at the time of his original illness was under the care of Dr. Hughlings Jackson, had pain of this kind without intermission for forty-five years, and when I last saw him before the "Blitz" it showed no sign of abating); (2) an excessive reaction to painful stimuli. There seems every reason to suppose that these two varieties are different degrees of the same disturbance and so they have been regarded by all who have written on the subject. In the former we must suppose that the causal discharges are spontaneous (if we admit that the pain is spontaneous), but in the latter a stimulus is required and then the resulting discharge is

excessive. The latter appears therefore to be due to a subliminal discharging state—a condition of instability in which the discharge is not actually spontaneous, but occurs as a greatly exaggerated response to any adequate stimulus.

Hyperalgesia Associated with Recovering Sensory Nerve.

I have referred more than once to this subliminal discharging state—a state in which discharge is not spontaneous but occurs as an exaggerated response to physiological stimuli. This seems to occur at different levels of the "pain system." I have also referred to spontaneous discharge phenomena in imperfectly recovered motor nerve. It is no part of my thesis to try to explain all the peculiar sensory phenomena that accompany the recovery of sensory nerves, but here is another condition in which we meet with painful over-reaction with an explosive quality, not dissimilar to that of the thalamic syndrome and we must see whether our hypothesis is applicable to it, particularly as the theory by which it has been explained for so many years is in process of dissolution. I had felt for a long time that Head's theory of a "protopathic" sensory system controlled by a biologically newer "epicritic" (Head, 1905), was, if nothing more, too complicated, and now that Dr. Walshe in his recent review has stated the objections to it in some detail, I need not recount them here (Walshe, 1942).

In trying to find an explanation for the sensations described or experienced, one starts *a priori* from the assumption that they must be due to events *directly* associated with the injured and regenerating structure, and the fact that no hyperalgesia or "intensification" of pain sensation accompanies recovery from mere functional block of the nerve, suggests strongly that the abnormal sensory phenomena are closely associated with actual regeneration: in the absence of regeneration (i.e. after nerve block with novocain) there is no over-reaction. Bearing in mind the phenomena of spontaneous discharge manifested by the imperfectly recovered facial nerve, I tentatively applied the hypothesis that the energy substance of the immature nerve was in an unstable state—as, for instance, incompletely synthesized—in other words, I supposed a subliminal discharging state. The development of this hypothesis is closely similar to that of Trotter's suggestion of imperfect insulation, involving a pathological reaction in the nerve tissue. Trotter's "pathological tissue reaction" must involve an expenditure of energy and, therefore, a breakdown of energy substance, and whether the excessive reaction is due to an instability of the energy substance or to abnormal conditions in which it finds itself is a question

which cannot be determined and which need not delay us. Suffice it to say that both variants seem to offer an explanation of the observed facts without requiring us to postulate a duplicate system of pain fibres and an elaborate system of control.

The responses to painful stimuli and to stroking with the finger dominate the observed disturbances until recovery is nearly complete, and this may be due to the greater number and earlier recovery of the fibres serving these modalities. Excessive explosiveness of the energy substance of the developing fibre accounts, I think, for the quality of the pain, but imperfect insulation of the fibres from each other may have to be invoked to account for the spread of sensation[1] which, in my experience, is considerable and produces a tingling effect. With approaching maturity the state of over-responsiveness to pain passes off and, at the same time, other modalities of sensation are restored owing (as I suppose) to recovery of their own fibres, but if full recovery fails to occur some degree of painful over-reaction may persist.

In the earlier weeks, that is, before there is any return of sensation attributable to regeneration, over-reaction or intensification of painful responses may occur in the marginal zone around the anæsthetic area. I used to suppose that this might be a consequence of a central excitatory state in the spinal cord associated with the activities of degeneration and regeneration of the affected nerve, but Weddell's recent work showing that pain fibres grow into the anæsthetic area from the surrounding areas at an early date provides the basis for another explanation, because if we attribute instability and over-reaction to the regenerating main nerve we must allow similar attributes during their immaturity to these invading filaments, and this may be the cause of the marginal hyperalgesia during their period of growth.

It is obvious that I cannot in a review of this kind pursue this large subject further and I must simply submit for your consideration the hypothesis of instability in a regenerating nerve as an explanation of some of the peculiar features that attach to the recovery of sensory nerves.

VI. Spontaneous Discharge in a Reflex Arc.

If in tabes spontaneous discharges occur in degenerating cells of the pain system, giving rise to lightning pains, is it likely that such discharges are limited to pain cells? What would happen if similar discharges

[1]The whole subject of the spread of abnormal excitations in different parts of the nervous system—cerebral cortex, thalamus, tracts and peripheral nerves—is one which calls for investigation and discussion.

occurred in cells which constituted the afferent portion of a reflex arc? One of my patients who was subject to tabetic pains in his thigh said that when the pains occurred the leg "kicked" and this I think was probably due to a reflexly excited contraction of the quadriceps, similar to the knee-jerk, but due to stimuli thrown into the reflex arc in association with the tabetic pain.

When I first asked myself what would be the effect of a discharging lesion involving a reflex arc, I soon realized that all the structures in which visceral crises occurred were governed by reflexes and that these reflexes were under normal conditions excited by irritants in the respective viscera. An irritant in the stomach excites a reflex which brings about vomiting. The reflex arc seems to pass by visceral afferent fibres to the mid-dorsal posterior spinal roots, then by ascending fibres in the anterior columns of the spinal cord to the vomiting centre in the medulla and this centre makes use of the vagus to excite anti-peristalsis. It is evident that vomiting would be excited equally if stimuli were thrown into the reflex arc as a result of spontaneous discharges from degenerating cells in the afferent nerves concerned. The effect would be not any isolated movement or twitching, but the whole co-ordinated group of movements that vomiting normally involves, and that is what occurs in the gastric crisis. The duration of a crisis is much the same as that of a bout of tabetic pains. Sir William Thorburn (1921) reported very good results in cases of frequent gastric crises from division of the 4th to the 8th dorsal posterior spinal roots on each side, and other surgeons have reported similar favourable effects both from this operation and from anterolateral cordotomy. Now-a-days, with the infrequency of tabes, visceral crises have become so uncommon that we have little opportunity to observe the outcome of such procedures.[1]

Rectal and vesical crises are subject to similar explanations, and, moreover, each of them is accompanied by a sensation as if an irritant were present in the viscus concerned.

Laryngeal crises involve the reflex which contracts the glottis when a foreign body enters it. Not merely the larynx is involved but the diaphragm as well and in fact the whole respiratory mechanism, so that here again there is a whole group of co-ordinated movements, such as we see in any complex reflex. Hughlings Jackson discussed the problem of laryngeal crises in almost the last of his "Neurological Fragments" (Jackson, 1925), and while he does not actually state that he considered that the

[1] I find that a similar explanation of gastric crises was proposed by Head and Fearnsides, 1914, *Brain*, **37**, 23.

primary abnormal discharge was in afferent elements he postulates an "irritative state," "a super-positive lesion" of sensory elements in the medulla, and he seems to have regarded that as the excitant of a spasm which constituted the first stage of the crisis. He describes crises which went on to convulsions as a result of asphyxiation.

It is certainly in keeping with the clinical features of tabes to postulate that spontaneous discharges occur in afferent elements, and spontaneous discharges in the afferent side of reflex arcs seem to offer a full explanation for these peculiar visceral attacks, for which no other adequate hypothesis has to my knowledge been proposed.

VII. Concluding Remarks.

Further Possible Applications.

It may seem to you that I apply this theory to a great many symptoms, but I assure you that it has further applications to which I have made no reference. For instance, I have said nothing of the more complex abnormal movements such as chorea, athetosis, and spasmodic torticollis, in all of which abnormal discharges must be concerned, although we know little of their sites. I may remind you also of the conclusion of that paragraph of Pasteur's from which I have already quoted: ". . . the characteristic of a true theory is its fruitfulness."

Differential Diagnosis.

A question which remains for discussion is whether it is possible to determine whether a given symptom of "positive" character associated with a lesion is due to spontaneous discharge from damaged elements or to the over-action of released centres. I doubt whether the point can always be decided, but, nevertheless, some general rules can be given. The phenomena of "spontaneous" discharge are in general of an "explosive," spasmodic, exaggerated, compelling character—fits, twitchings, the spasms of tetanus, visceral crises, the pains of trigeminal neuralgia and of tabes, the "explosive" exaggerated response to pin-prick in the thalamic syndrome or its insistent spontaneous pain. The overaction of lower centres is of a more continuous and gentler nature. Again, many (but not all) phenomena of spontaneous discharge occur equally during sleep and in the waking state. Release phenomena if they involve abnormal movements seem to cease during sleep. This is a very rough and ready and imperfect generalization, but present knowledge does not permit us to go further.

Conclusion.

I may conclude as I began, by saying again that the hypothesis of spontaneous discharge from nerve tissue under abnormal conditions has a much wider application in the theory of clinical neurology than has hitherto been accorded to it. This theory represents a point of view, and it is a point of view which is helpful to the clinician: it provides explanations for, and enables us to see a certain unity in, a number of symptoms for which the current explanations are unsatisfactory.

I may perhaps go further and say that the nerve cell possesses for the clinician a property which the physiologist is just beginning to discover. The physiologist, dealing with the normal, is concerned with the nerve cells two fundamental properties of excitability and conductivity, and the impulses aroused in it when it is stimulated and even the sources of its energy. But for the clinician, dealing with morbid states, the cell has this third property—this liability to emit impulses under abnormal conditions without a physiological stimulus, the impulses then being irregular and usually excessive. Now that Adrian has observed and recorded such impulses in the physiological laboratory and so brought this property of spontaneous discharge under conditions of experimental study we can hope for more elucidation of it.

SUMMARY.

Hughlings Jackson's theory of the "discharging lesion" is capable of a wider application in clinical neurology than has hitherto been accorded to it. The theory postulated (1) that nervous tissue "stored up" energy by means of a chemical energy substance and under the influence of physiological stimuli some of this energy was emitted in an orderly and controlled manner, and (2) that under certain abnormal conditions the energy substance might break down causing discharge of energy without a physiological stimulus, and then the discharge was excessive, uncontrolled and disorderly.

Modern physiological knowledge lends support to this theory, especially by the analogy of the physiology of the muscle cell, and by the discovery of the injury current in nerve.

Discharges need not be "explosive"; many are better described as spluttering and others are of a continuous character.

Phenomena of abnormal discharge are not necessarily transient. Some, such as trismus, may be continuous for a long time, and others of the spluttering type, such as peripheral facial spasm, may persist for the rest of the patient's life. In such instances we must suppose that in the discharg-

ing nervous elements the store of energy on which the discharge depends is being renewed as quickly as it is being expended.

The theory is applied to the thalamic syndrome, this syndrome being attributed to a discharging lesion in the thalamus constituted by cells which are imperfectly recovered after a vascular lesion.

In a recovering sensory nerve a state of instability may be the basis of that hyperalgesia which Head attributed to "protopathic sensation."

Abnormal discharges may occur in the afferent elements of a reflex-arc, and such may be responsible for the visceral crises of tabes.

Symptoms due to discharging lesions have, in general, an excessive, "explosive" and compelling quality.

The theory provides explanations for, and enables us to see a certain unity in, a number of symptoms for which the current explanations are unsatisfactory.

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A CASE OF TOPOGRAPHICAL DISORIENTATION ASSOCIATED WITH A UNILATERAL CEREBRAL LESION.

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I. Introduction.

Three cases of cerebral lesion characterized by profound disorders of spatial orientation and topographical memory were described by O. Meyer in 1900. In these cases, unlike those described some years later by Holmes (1918, 1919), there was no visual disorientation in the sense of inability to appreciate direction or distance in the central field of vision. Nor was there any inability to recognize common objects (visual object agnosia). But Meyer's patients had the utmost difficulty in orienting themselves in their accustomed surroundings and in following (or describing) long-familiar routes. The disorder thus appeared to bear essentially on *topographical memory* and the complex functions of place-recognition, route-finding and spatial recall which it normally makes possible. The lesion responsible was right-sided in the first case, left-sided in the second and bilateral in the third.

The peculiar difficulties in recognizing places exhibited by all three cases were graphically described by Meyer (1900) in his first patient. This was a man of 49 who sustained a vascular lesion without loss of consciousness but with temporary loss of vision. Vision in the right half-field was rapidly restored but on admission to Meyer's clinic on the following day the patient displayed a left homonymous hemianopia (complete apart from 2 degrees central sparing) together with a gross disturbance of spatial orientation. Examination revealed no aphasia or object-agnosia and no obvious disorder of depth and distance perception in central vision (the patient could always avoid successfully obstacles scattered in his path). It was further found that the patient could draw common objects from memory but, in reading, he showed some tendency to lose the place and failed to follow the lines in an orderly manner. General memory was good for remote events but slightly impaired for those that had taken place more recently. Memory for topographical relationships was grossly impaired although geographical data *as such* could be recalled correctly. Thus the patient could always name correctly the streets in which the principal public buildings of Breslau (his home

town) were situated and in some cases could even recall their street-numbers. But he was utterly unable to describe, draw or represent in any way how he would reach these buildings from his home (or from the clinic) and completely failed to orient himself on a map of Breslau.

The difficulties of spatial orientation were even more clearly revealed on a practical test. On one occasion, for example, the patient was asked by Meyer to lead the way from the clinic to his home. When led out of the clinic (which he had formerly known well), the patient was very uncertain as to his immediate whereabouts and the direction which should be taken. He eventually recognized the sign on a neighbouring shop and this gave him a measure of orientation. Very soon, however, the patient once again became disoriented and on reaching the main square in Breslau anxiously sought the placards exhibiting the names of the various streets. Eventually, he discovered his precise whereabouts by inference from the numbers of passing street-cars. He then directed his steps correctly with reference to his home but was much aided by following the car-lines. At each successive crossing, however, his uncertainty was renewed and he was reassured only when he happened to catch sight of the name (or sign) on a shop which he had formerly patronized. Retaining, as he did, the knowledge of the name of the street in which the shop in question was situated, he was able to deduce his immediate whereabouts in a cumbrous though accurate manner. Orientation was thus achieved by deduction rather than by true recognition. In this manner, slowly and laboriously, the patient ultimately found his way to his home.

Meyer adds that this patient had comparable, and perhaps even grosser, troubles of orientation and route-finding in the clinic itself. Thus he never learnt with certainty whether he should proceed up or downstairs from the room in which he was habitually examined in order to reach his ward. The latter, moreover, was identified as a rule by the fact that his fellow-patient in the ward wore a conspicuous black beard. If this patient happened to be absent, recognition was generally achieved by reference to other individual objects, e.g. the bell at the bedside. Meyer found that the patient's recognition of a room was always effected by reference to small and conspicuous objects and could be readily upset if these were hidden or removed. In general, recognition of an extended spatial setting in an immediate manner appeared beyond the patient's capacity.

Lange (1936, pp. 854-5), in a review of this case, points out that the patient's memory was intact in so far as *chronological sequence* is concerned but grossly defective from the point of view of *spatial articulation*. He argues that the disturbance shown here in the field of memory is

closely analogous to the better-known disturbances in the sphere of visual space perception often classified as the *visual-spatial agnosias* (cf. Poetzl, 1928: Paterson and Zangwill, 1944*b*). In disorders of this type, there is no inability to recognize objects as such but their inter-relationship in terms of the wider spatial setting is appreciably disturbed. In Meyer's cases, equally, there is no inability to represent objects as such but they cannot be inter-related from the point of view of coherent topographical representation. Lange therefore argues that the general principles of spatial organization which hold good for visual perception likewise hold good for topographical memory, but, from the point of view of *breakdown*, perception and memory are to some extent at least independent. Thus by no means all cases of visual-spatial agnosia show disturbances of topographical memory, and, conversely, visual perception (in the narrower sense at least) may be largely intact in cases such as those of Meyer in which the topographical amnesia is pronounced.[1] On the whole, the available evidence suggests that disorders of topographical memory proper, in cases of unilateral lesion at least, are relatively less common than disorders of spatial organization in its more narrowly perceptual aspects.

It appears probable that *bilateral* occipito-parietal lesions of the type giving rise to complete visual disorientation (Best, 1917; Holmes, 1918, 1919) invariably give rise to an additional disorder of topographical memory. At all events, a disorder in this field as especially stressed by Holmes (1918, 1919) in his reports of these cases and was studied in detail in one (Holmes and Horrax, 1919). In none of Holmes's cases, however, was the recognition of places well known to the patient before sustaining his injury studied in detail, doubtless because the prevailing war conditions did not permit practical studies of orientation comparable to those of Meyer (1900). But had these been undertaken, there can be

[1]It is important in this connection to distinguish between recognition or recall of spatial settings familiar to the patient before sustaining his lesion and his behaviour in regard to those with which he may be called upon to deal during and after his illness and with which he was not previously acquainted. In a large number of the cases of visual-spatial agnosia described in the literature gross disorders of recognition and route-finding *in the hospital setting* have been reported although the patient's memory for spatial settings *dating from before the illness* appeared to be intact. The cases described by Poetzl (1928), Scheller and Seidemann (1932), Russell Brain (1941), and Paterson and Zangwill (1944*b*) are of this type. We shall therefore use the term *topographical memory defect* with exclusive reference to cases such as those of Meyer (1900) in which there are disorders of orientation, recognition and recall bearing on settings with which the patients were well acquainted previous to the illness. Cobb (1944, p. 214) refers to lesions of areas 18 and 19 as being especially prone to give rise to topographical disturbances of this type.

little doubt that very gross disorders of recognition and route-finding would have been observed. As regards the incidence of disorders of this type in cases of *unilateral* lesion, it will be recalled that the lesion was (at all events predominantly) right-sided in one of Meyer's cases and left-sided in a second. We may also comment that a case of right-sided lesion with somewhat comparable disorders of route-finding and topographical recall was reported as long ago as 1876 by Hughlings Jackson. This case will be considered in greater detail at a later stage. In the more recent literature, Nielsen and his associates have called particular attention to topographical memory disorders in cases of unilateral cerebral lesion (Nielsen, 1940; Nielsen and Raney, 1940). These authors hold the view that the concept of cerebral dominance can justifiably be extended to cover functions of this general type. Topographical memory, on this view, is subserved, along with visual object-recognition, by the "dominant" occipital lobe. This is usually the left, but in some cases Nielsen believes that "crossed temporo-occipital dominance" may occur, with the result that a right-sided temporo-occipital lesion in a right-handed patient may give rise to visual agnosia and topographical memory loss without aphasia (*see also* Russell Brain, 1941). We may comment that whereas the evidence regarding determination of visual object-agnosia by lesions of the dominant hemisphere is reasonably good (Nielsen, 1937), the evidence regarding a differential effect of the major and minor hemisphere in conditioning disorders in the visual-spatial sphere appears, to the present writers at least, distinctly inconclusive. In the first place, the balance of evidence in the spatial-agnostic disturbances suggests that the symptoms following a parieto-occipital lesion of either hemisphere are very similar and perhaps identical. As the present writers have stated elsewhere (Paterson and Zangwill, 1944*b*), there appear to be no clear-cut disturbances of visual space perception associated with lesions of the right cerebral hemisphere which are not also found with corresponding lesions of the left side. And in the second place, cases with gross disorder of topographical memory associated with unilateral lesions have been rather seldom reported in the literature and the evidence available for assessing Nielsen's theory is in consequence limited. Further, as we have said, visual object-agnosia was not present in the cases reported by Meyer (1900). It therefore appears to us premature to enter into discussions of cerebral dominance in the general field of visual space perception and topographical memory.

It may justifiably be asked whether topographical memory loss ever occurs as an isolated syndrome unaccompanied by disorders in other fields. In Meyer's cases, as we have seen, the disorientation was in the foreground

of the clinical picture but all three patients gave evidence of a more widespread disturbance. Hemianopia was present in all cases and disorders of oculomotor sequence and control in at least one. There was some degree of memory defect for recent events in two cases; in one of which, indeed, the clinical picture was at first that of the amnesic syndrome. At the same time, it is important to bear in mind that all these defects are commonly found in cases without topographical memory loss and that this function may be largely intact in patients with various types of perceptual disability. As Russell Brain (1941) points out, a case of visual disorientation (with or without left-sided visual agnosia) may retain the capacity to describe oft-traversed routes and scenes with which the patient was acquainted before his illness. "Loss of this ability," he concludes, "is a disorder independent both of the other forms of visual disorientation and loss of memory for objects" (*Brain*, 1941, p. 270). The upshot appears to be that although topographical memory loss is commonly, perhaps invariably, associated with visual disorientation in cases of bilateral lesion, and is occasionally associated with both visual object-agnosia and the various types of visual-spatial disability which may be associated with unilateral lesions, it enjoys a certain functional independence of all these types of disorder.

The object of the present paper is to report a case in which disturbances of topographical memory were strikingly in evidence and were made the subject of a detailed study. We do not pretend that the case was other than complicated and indeed a variety of mental symptoms were in evidence when the patient first came under our observation. Included in the picture at this time was a general memory disturbance of Korsakoff type, a left homonymous hemianopia with associated neglect of the left half of visual space and the left upper extremity (autotopagnosia), spatial-agnostic symptoms with apraxia for dressing, and some degree of personality change with notable lack of insight into all disabilities. Apart from the hemianopia and the associated left-sided neglect, however, all these disabilities recovered very much more rapidly than the topographical memory disturbance, which was indeed grossly in evidence one year after the patient's admission to the Brain Injuries Unit. At this time, in fact, he presented a picture virtually identical with the case described by Meyer (1900) and summarized above. In view first of the unilateral and well-circumscribed character of the lesion; secondly, of the absence of aphasia; and thirdly, of the unusually favourable opportunities for undertaking detailed practical tests of orientation, it is thought that the case is worthy of being placed on record.

II. Case Report.

The patient was a healthy married man, aged 34, who had been formerly the manager of a small insurance concern. He sustained a penetrating head wound of the right parietal area.

The missile, an irregular mortar fragment roughly 2 × 1 × 1 cm., penetrated the right upper parietal lobe and passed downwards, medially and backwards, coming to rest in the angle between the falx and the tentorium and not crossing the mid-line (*see* X-rays of skull in fig. 1). The fact that the missile came to rest in this position suggests that it had been travelling at a comparatively low velocity. Patient was rendered unconscious. An hour or so later, was described as "mentally unresponsive." A few hours later, was restless, had a flaccid hemiparesis of the left side, responded to noxious stimuli on the right side but not on the left. Had a right facial palsy, the left eye tended to divergent strabismus. Right pupil was larger than left; both responded to light. There was bleeding from right ear. The patient could be roused by repeated questioning but was incoherent in his replies. On following day, operation performed: Bone opening enlarged and metal foreign body in angle between falx and tentorium removed by magnet. Brain track 3 in. long was cleared and penicillin tube inserted. Cerebral œdema was present. The next day patient was speaking better; general condition was good but hemiparesis still in evidence. Two days later, was described as talking "well and rationally." Was moving left leg well but left arm still paretic. Had tic of drumming right leg on bed.

Five days later, speech was good but the patient was observed to be grossly disoriented. Improvement in general condition was rapid but disorientation remained unchanged. Was transferred to this country where he was found to be "irrational" and denied that he had been injured in any way. He said he used to be "nervous" but was able to control himself much better in the past few months. Neurological examination showed pupils reacting normally to light and slight left hemiparesis. Was described as euphoric and completely disoriented.

The patient was admitted to the Brain Injuries Unit seven weeks after sustaining his injury.

Neurological re-examination.—Right-handed man, no aphasia. Visual acuity J1 in both eyes; visual fields to confrontation showed left homonymous hemianopia with no central sparing. Fundi normal. Left pupil was slightly larger than right: both reacting sluggishly to light but well to convergence. Marked variation in size of pupils—sometimes considerably dilated. Definite right lower facial weakness noticed. Other cranial nerves intact. Slight increase of tone in left limbs, but a trace of weakness only in the left upper limb. No loss of power elsewhere. Fine movements in fingers of the left hand were rather impaired. Tendon reflexes slightly brisker in the left upper limbs than in right; knee-jerks were brisk; ankle-jerk on left side slightly increased. Considerable impairment of proprioceptive sensation in the left upper limb, especially in the joints of the fingers and hand. No loss on right side or in lower limbs. Exteroceptive sensation was slightly depressed for all modalities in left upper limb, upper part of trunk, and neck, but not in face. Patient complained of gross tenderness and pain on movement over the 3rd and 4th lumbar spinous processes, but no objective findings in this respect apart from some restriction of movement of spine in that region. Visual fields were charted a few days later and showed left homonymous hemianopia, complete apart from 2 degrees central sparing.

The patient made a good superficial impression on *psychiatric interview*. His social behaviour was correct and conversation well maintained. His views on topics of general and current interest were expressed forcibly and well. But closer observation revealed gross disturbances of higher mental function. These comprised (1) an amnesic syndrome of Korsakoff type; (2) neglect of the left upper extremity (autotopagnosia) with an associated agnosia for the left half of external visual space; (3) marked visual-constructive disability of parietal type; (4) apraxia for dressing (Brain, 1941); and (5) outspoken loss of spontaneity and insight. The disorder of topographical orientation and memory which forms the principal subject of this paper was studied for the most part at a later stage. In view of the complexity of the case, however, it is perhaps advisable to give some account of the neuropsychiatric findings on early examination.

(1) *Amnesic syndrome.*—The patient could at first give no coherent information as to his recent movements but was aware that he was in hospital near Edinburgh. When taken by wheeled chair to an adjacent room and questioned as to his whereabouts he was prone to say that he was now in another hospital to which he had been brought by ambulance or car (confabulation). He grossly over-estimated this "journey," often transforming the distance into a matter of miles and the time taken into a matter of hours. Though recognizing his doctors from day to day, the patient always said that it was a matter of weeks (or even months) since he had last seen a given member of the staff. (The actual interval was seldom more than a day.) This tendency to *over-estimate grossly the passage of time* was pronounced and consistent during the patient's first weeks in hospital and applied equally to very short intervals. Thus on one test he estimated exactly one minute as a quarter of an hour and an interval of fifteen minutes as an hour or two.

The patient's memory for ordinary events in hospital routine was very defective during the first few weeks after admission. It was found that outstanding events (e.g. a visit to a music hall in Edinburgh) were adequately recalled after several days but the temporal and spatial reference was grossly defective. Over-estimation of time and some degree of fabrication were rather marked. Formal *tests of retention* showed that immediate memory was only slightly affected and predominantly for visual material. Thus the patient could repeat 8 digits forwards without error but could enumerate only 2 of a series of 5 pictures of simple common objects from immediate memory. Elements of short stories could be recalled with prompting after several days but there was some fabrication. Experiments on *recognition* showed rather marked defects in the case of visual material (pictures, &c.). Often he said that a picture shown for the second time after a few moments was not precisely identical with the one shown earlier and was rejected as unfamiliar. Thus, on one occasion, the patient refused to accept as his own handiwork a bird which he had sketched a few minutes before on the ground that he had not put in the eye in his drawing. These errors of recognition are reminiscent of the phenomena described by one of us (Zangwill, 1941) in cases of alcoholic Korsakoff psychosis.

The patient's *orientation* was at first grossly defective both for place and time. As regards *place*, he always gave the name of the hospital correctly but tended to situate the latter much closer to his home in Edinburgh than was actually the case. One may note that the location of the hospital had been well known to him

before his injury. He was apt to misinterpret the hospital buildings as being in the close vicinity of his home and stated that he could reach the centre of Edinburgh on foot in a matter of twenty minutes. When the discrepancies between his present statements and his earlier knowledge of the relation of the hospital to his home were pointed out to him, he rationalized in the evasive manner to which attention has been called by the present writers in an earlier discussion of orientation in the confusional state (Paterson and Zangwill, 1944*a*). As regards *time*, the patient was oriented throughout for year but generally gave the month (actually July) as November or December. His orientation for time of day was at first excessively poor, and, as we shall mention later, no use was made of the wrist watch which the patient always wore on his left wrist.

There was at first a considerable *retrograde amnesia* of which the scope could not be precisely defined. The patient could narrate the main events of his earlier life but was very prone to dislocate the order and sequence of the principal events and experiences. He was distinctly hazy as to his movements during the few months previous to his injury and tended to fabricate when questions were pressed.

Insight into the memory defect was at first completely lacking, and, with recovery, was in retrospect only partial. Orientation and memory in all fields gradually improved and on discharge from hospital three and a half months after admission the patient was fully oriented and able to recall very adequately the main events of day-to-day life in hospital. His improvement was also shown on standard retention tests although some residual retention defect for visual material was in evidence one year after discharge.

(2) *Left-sided agnostic disorders.*—It was noticed early that the patient *totally neglected his left upper extremity* and made virtually no use of it despite good preservation of motor power. The left hand had literally "dropped out of the patient's spontaneity" (Nielsen, 1937) and its spontaneous use was first noted only a month after admission. Thereafter its use became progressively more frequent and he began to refer to his wrist watch when asked the time. Nevertheless, a marked reluctance to use the left hand if it could possibly be avoided was noted throughout the patient's stay in hospital, and, if he were carrying something in it, he might commonly forget to lay it down when the proper occasion arose.

A corresponding neglect of the *left half of visual space* was also observed. The patient tended to deviate to the right in walking and consistently failed to appreciate doors and turnings on his left-hand side even when he was aware of their presence. Thus he would automatically turn to the right whenever the opportunity afforded and consequently tended to wander in a circle. The disorder was thus very similar to the cases analysed by Russell Brain (1941). It was also noticed that the patient neglected the left-hand side of a picture or the left-hand page of a book despite the fact that his attention was constantly being drawn to the oversight. This brings out well the amnesic element in the syndrome. In playing draughts, it was observed that the patient neglected the left side of the board and when his attention was drawn to the pieces on this side he recognized them but immediately thereafter forgot them. This disability likewise improved gradually, but a preference for right-hand turnings was still in evidence on discharge.

(3) *Visual-constructive disability.*—The patient displayed very marked impairment in the field of visual perception in its higher spatial aspects and in all constructional activity normally carried out under visual control. The picture was essentially that described by the present writers in an earlier communication (Paterson and Zangwill, 1944*b*) and only the barest summary of our findings in the present case will therefore be given here. In the first place, there was probably

some slight degree of impairment of oculomotor co-ordination, as shown by the patient's difficulty in counting scattered objects—even those to the right of the fixation point. The sequence of eye-movements in reading, on the other hand, appeared essentially intact. In the second place, there were gross disorders in copying, drawing, arranging mosaics and in all types of constructional work. In free-hand drawing, the patient always began on the right-hand side and often omitted the left half of the object he was attempting to draw. On occasion, however, he reduplicated a part on the left hand side which he had already drawn a few moments earlier. He failed on the simplest types of "performance test" (Paterson and Zangwill, 1944*b*) and on a very simple weaving task in Occupational Therapy which demanded only the threading of a thong alternately over and under a fixed framework. Improvement on these tasks was noted to a limited extent but specific re-educational exercises were of no avail.

(4) *Apraxia for dressing.*—Dressing in the ordinary way was bad, the patient being more than once observed to have on his trousers back to front. He regularly prevailed on his fellow-patients to tie his tie for him. On one occasion, we called the patient's attention to an error of dress and he removed his trousers in order to replace them correctly This, however, he forgot to do until reminded. He showed the greatest difficulty in orientation, at first trying to put on the trousers inside-out. Eventually, he got the right trouser-leg turned right way out and introduced his leg into it correctly but the left trouser-leg remained inside-out. He tried nevertheless to get his left leg into the trousers with understandable lack of success. At one point, he appeared to "lose" the left trouser-leg completely and endeavoured to introduce both legs into the right one. Apropos of this situation, he jocularly remarked: "Should be coming into it now unless it's something the Jews made!" These difficulties in dressing could be regularly provoked and the patient's activity in this respect was in striking contrast to his habitual lethargy. Comparable difficulties of dressing in patients with right-sided cerebral lesions have been described by Hughlings Jackson (1876) and Russell Brain (1941).

(5) *Spontaneity and insight.*—There was at first a marked *lack of spontaneity* except in situations in which a specific response was demanded (as on the constructional and dressing tests). The patient was content to lie in bed and smoke cigarettes if they were available. He would, however, respond promptly to questions and co-operated fully on all the special investigations. But he rarely took the initiative in conversation except to detail complaints regarding his back. He believed that he had sustained an operation on the latter and that several vertebræ had been removed. He further alleged that the pain was exacerbated by nursing mismanagement. These complaints, at first truly delusional in status, gradually became less frequent and ultimately receded.

Mood was on the whole equable if somewhat apathetic. When attention was drawn to his back, however, the patient was at once roused and repeated in an angry tone of voice that he had been mishandled. In conversation, he became evasive and at times oppositional if the question of insight was touched upon. The patient developed a peculiar sudden *laugh* with which he constantly ended his remarks. Although this response put one in mind of a "catastrophic reaction" (Goldstein, 1939), it is possible that it represented an automatic "false laughter" response of subcortical type.

The patient was friendly and co-operative in the ward and mixed well with his fellow-patients. On visits from his relatives, however, he was said to be more subdued and unresponsive than he had been previously. Although tending to be discursive and repetitive in conversation, his *judgment* on matters other than those pertaining to his immediate condition and situation appeared adequate.

PLATE X.

FIG. 1 (*b*)

FIG. 1 (*a*)

To illustrate article by Andrew Paterson and O. L. Zangwill.

PLATE XI.

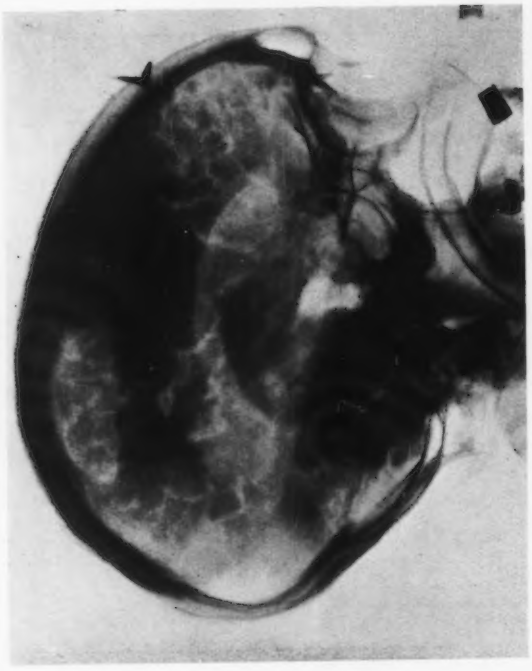

FIG. 2 (*b*)

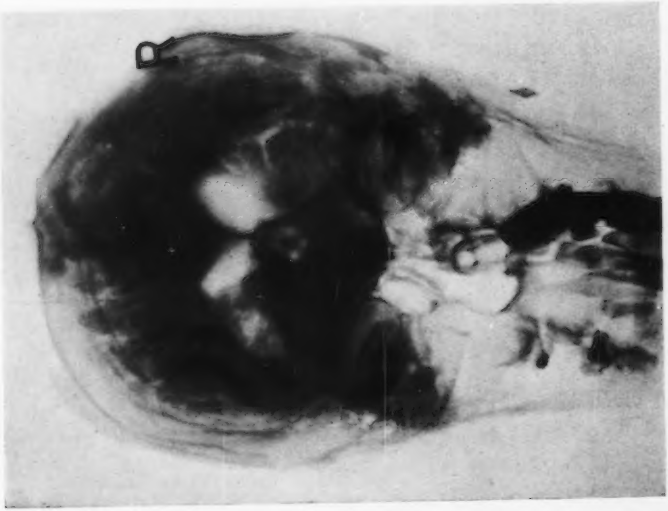

FIG. 2 (*a*)

To illustrate article by Andrew Paterson and O. L. Zangwill.

Routine *psychological examination* suggested that the patient had been slightly *above average* in general intelligence and had sustained no general intellectual deterioration in the sense of post-traumatic dementia.

The level obtained on *verbal intelligence tests* was very satisfactory. On the Terman Vocabulary (Terman, 1919), the score was 58. On the Bellevue Verbal Intelligence-Scale (Wechsler, 1941), an I.Q. of 108 was obtained. There was no significant variability in score on the five subtests composing this battery. The Weigl Sorting Test (Goldstein and Scheerer, 1941) was performed in a normal manner. The patient was of course grossly impaired on visual or performance tests of intelligence and obtained a score of only 26 on Raven's Matrices (Raven, 1938).

The patient's *condition on discharge* was as follows: Physical condition was now very satisfactory and residual neurological findings comprised only the complete left homonymous hemianopia and a slight impairment of proprioceptive sensation in the fingers of the left hand. Gait was normal and displayed the usual free associated movements of the arms. From the neuropsychiatric point of view, remaining disabilities were (in addition to the orientation disturbance discussed below) neglect of the left half of visual space with tendency to turn right in route-finding, high-grade spatial-agnostic and constructive apraxic disorders, some memory-retention defect for visual material, some degree of personality-change and marked lack of insight generally. The patient was keen to return to work and did in fact do so shortly after his return home. It was reported by relatives that he was very slow in getting from his house to his place of business (doubtless on account of the route-finding and topographic memory disabilities) and it was considered that his practical judgment in business matters was somewhat impaired.

Air-encephalography was undertaken shortly before the patient's discharge and posterior and lateral views are shown in fig. 2. The outstanding feature is the gross dilatation of the temporal horn of the right lateral ventricle at its junction with the body of the ventricle. The whole ventricular system is displaced towards the right-hand side and there is a compensatory dilatation of the left lateral and third ventricle. The gross loss of brain substance in the posterior part of the right temporal lobe indicated by the encephalograms suggests that a hæmorrhage occurred in this area with heavy destruction of the surrounding brain tissue. General contusional damage may reasonably be considered to be small in view of the short duration of unconsciousness and the fact that the missile came to rest in relatively soft tissue. Further evidence for this is also given by the encephalographic findings.

The patient *was re-examined* at intervals during the following year.

The neurological findings described above remained in evidence but the general condition and spontaneity continued to improve.

III. Analysis of Topographical Orientation.

(1) *Orientation in Hospital.*

It has been mentioned above that the patient was at first disoriented as to *place* in the sense that he located the hospital (previously well known to him) in the close vicinity of his own home several miles distant. In view of the fact that this phenomenon of *spatial displacement* is not uncommon in the post-traumatic confusional state (Paterson and Zangwill, 1944*a*) it will not be further discussed here. It has also been mentioned that the patient consistently turned to the right in endeavouring to find his way about the hospital and this made it virtually impossible for him to traverse the wards and hospital grounds unaccompanied. Over and above these defects, however, the patient displayed a very marked *inability to recognize rooms and buildings generally*. Although his recognition of any type of visual material (e.g. small objects or pictures) was at first defective and paramnesic, this improved very much more rapidly than his ability to identify individual rooms in which he was frequently interviewed.

Analysis of recognition defect.—His ability to recognize rooms appeared to depend, as in Meyer's patient (Meyer, 1900), on the presence of conspicuous individual detail. Thus recognition of a room in which he was regularly interviewed was found to depend on a small notice labelled "A.C.Plugs." If this notice was concealed recognition at once broke down. The patient might then deny that he had ever been in the room in question although admitting that he was acquainted with similar rooms. Rooms presenting few outstanding details were seldom recognized despite considerable familiarity with them. The patient's recognition of the individual buildings of which the hospital is composed was similarly defective and mistakes were often made.

There was very little improvement in the patient's ability to recognize rooms and buildings or to find his way around the hospital during the three months' period of observation.

(2) *Orientation in Habitual Surroundings.*

The patient's orientation in *Edinburgh* (his home town) and his recognition of prominent buildings and landmarks in this city was studied in some detail over a period of nine months. On his first visit home, two months after admission, he was accompanied by another patient, reliable in character, who furnished us with a detailed report of our patient's behaviour on the following day. Extracts from this may be quoted:—

On arrival in Edinburgh, the patient failed to recognize the proper point at which he should alight from the bus in order to reach his home most conveniently and was carried on to the terminus in the centre of the city. Here, he was obliged to ask a passer-by how to reach his home. We may remark that the bus-terminus is adjacent to the patient's former place of business and was very well known to him in the past. The patient and his escort then boarded the appropriate tram-car (whose number the patient retained) and travelled to the terminus which happened to be the stop required. From here, the patient correctly turned right into the street in which his house is situated but *failed to recognize the latter on passing it despite the fact that it lay to his right.* He turned back only when informed by his escort that they had already passed the required number.

Some days later, we ourselves drove the patient from the hospital into Edinburgh and questioned him repeatedly as to his whereabouts during the drive. A record of his responses was made on the spot and some extracts may be given here:—

We stopped first at a point of vantage, about a mile from the outskirts of Edinburgh, and commanding an extensive view of the city and its environs. An adjacent aerodrome, the only one in the immediate vicinity of the city, was identified correctly by the patient but a neighbouring hill he identified as one in fact on the opposite side of the city some eight miles distant from the aerodrome. On passing through the outer suburbs of Edinburgh, the patient was asked which part of the city he was now traversing. (This road was well known to the patient in the past and he had often driven through this part of Edinburgh before the war.) The answers given clearly showed that the patient was quite unable to orient himself correctly and he tended to give the names of districts lying between the centre of the city and his own home in fact several miles distant. He spontaneously recognized that the numbers on passing tram-cars were inconsistent with the statements that he had made regarding his whereabouts and thereby achieved a measure of orientation. Even so, he failed to recognize outstanding buildings (e.g. the Zoo) unless he happened to catch sight of a placard bearing its name. The distinctive outline of Arthur's Seat (a universally known hill and landmark in Edinburgh) was not recognized, the patient hazarding that the hill in question formed part of the Pentland Hills which lie some miles to the south of the city.

We drove slowly through the centre of the city, often stopping to ask the patient to identify prominent streets and buildings. The patient was quite clear as to his whereabouts, and named several streets correctly, but made the grossest errors in identifying individual buildings. Thus he entirely failed to recognize a large and prominent church at which he had been for many years a regular attendant. He recognized that it was in fact a church but stated that it was unfamiliar to him. When told its name, the patient rationalized his failure by alleging that it had been painted differently in the past (in fact there had been no change whatsoever in its external appearance). In the same way, the patient failed to recognize a prominent hotel with a distinctive tower and suggested that it might be St. Giles' Cathedral, in fact some distance away and out of sight. The well-known Scott Monument, when pointed out to him carefully from an exceptionally advantageous viewpoint, the patient thought might be the tower of the above-mentioned hotel.

On driving the patient to his home, he betrayed similar difficulties of orientation in regard to the various parts of the city traversed but recognized his own street as we entered it. On slowly traversing it, and instructing the patient to

signal when we reached his house, it was found that the patient was unable to recognize the latter as he passed it despite the fact that it lay on his right-hand side and that he was actively on the look-out for it.

A month later, we again drove the patient from the hospital into Edinburgh on the occasion of his final discharge. The patient's general memory had by this time very largely recovered and his difficulties in visual perception in the narrower sense had considerably receded. But no change in the patient's ability to orient himself precisely in the city could be detected. In traversing the outer suburbs, it was found that the patient was quite unable to maintain a consistent orientation despite the fact that at times he grasped his whereabouts correctly. At times he appeared to reply to our questions almost at hazard, mentioning in the course of a few minutes a number of districts remote both from his present location and from one another.

Our *follow-up studies* during the following year showed that the patient slowly regained some measure of orientation in the parts of the city which he had occasion to traverse to and from work and was relatively soon able to undertake the journey by tram-car unaccompanied. It was, however, reported that the time taken was excessive. It was also reported that his recognition of individual streets and buildings remained much impaired and the patient frequently named wrongly parts of the town traversed on his way to and from his place of business.

We undertook one further practical test of orientation in Edinburgh almost exactly one year after the patient's discharge from hospital. At this time, as we have said, the patient's general condition was satisfactory and his mental state further improved. Very considerable disturbance of orientation, route-finding and place recognition was, however, still in evidence.

The patient was driven slowly from the Royal Infirmary (which he recognized) through the central districts of the city. The first important road-crossing, known to him since childhood, was correctly recognized but the patient was unable to give consistent information regarding the individual streets converging on this junction or to specify the parts of the town to which they led. He recognized without difficulty the church which he had attended before the war (on which he failed on the earlier test) but completely failed to identify a neighbouring church, suggesting that it might be one in a district through which we had already passed and which was now some two miles from our present location. He also misinterpreted the next few streets through which we passed, suggesting that they were in the neighbourhood of the latter church. But on reaching the central district of Edinburgh orientation was temporarily restored. This, however, again broke down as we turned into the adjacent side-streets. On passing through a well-known street, the patient recognized at a distance a building with prominent red towers and deduced that we must therefore be approaching the street that

we were in fact traversing. It may be noted that although this building was adjacent to the patient's place of business, he would normally approach it from the opposite direction on his way to work.

The patient had by now some degree of insight into his orientation difficulties but did not appreciate their full gravity. He reported spontaneously that he paid greater attention to the details of streets and buildings than he had done formerly and agreed that these might assist him in ascertaining his whereabouts. But he denied being seriously handicapped in finding his way around in Edinburgh.

(3) *Plan Drawing.*

The patient was often requested to attempt a free-hand drawing of the *ground-plan* of his house or a *street-plan* of the central district of Edinburgh. These performances were grossly disturbed and suggested some degree of topographical memory loss in addition to the perceptual and constructive disabilities.

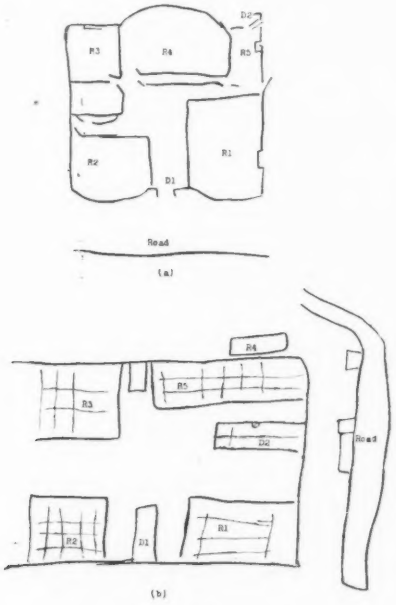

FIG. 3

Plan drawing.—One of the patient's attempts to draw the ground-plan of his house, obtained some four months after admission, is shown in fig. 3 (*b*) and may be compared with the plan (fig. 3 (*a*)) kindly drawn for us by the patient's wife.

The patient first blocked out three sides of the plan (omitting the left) and then introduced, one by one, the four separate rooms (R1—4) shown in the drawing. The doors (D1, D2) were inserted to our request, and it will be noted that they are apparently drawn in the frontal elevation. When asked about the blank spaces between the rooms, the patient said that they formed the hall The outside road, correctly represented in fig. 3 (*a*), is shown on the right-hand side of the plan parallel to the side of the building.

This performance, though betraying some memory-knowledge of the lay-out of the house, clearly brings out the effect of the left-sided spatial agnosia in addition to the specific difficulties of perceptual orientation and plan drawing to which we have called attention in an earlier communication (Paterson and Zangwill, 1944*b*).

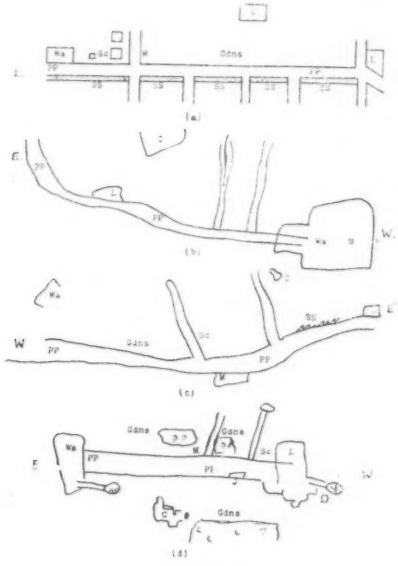

FIG. 4

Map drawing.—Three successive drawings made by the patient of Princes Street, Edinburgh's central thoroughfare, are shown in fig. 4 (*b*, *c*, and *d*) and may be compared with a corresponding plan (fig. 4 (*a*)) drawn by ourselves. On the first attempt (fig. 4 (*b*)), made seven weeks after admission, the patient began at the right-hand side of the paper and drew the uneven lines (PP) to represent the street itself. He labelled the right-hand end correctly as the West End, but drew in here the Waverley Station (W) and the Mound (M) which in fact lie at the opposite end of the street. Two of the main streets leading off Princes Street were interchanged and drawn on the same side as Edinburgh Castle (C). This would appear a very gross error which cannot be explained by transposition of left and right.

On the second attempt (fig. 4 (*c*)), made two weeks later, the patient again drew the street (PP) from right to left and again interchanged its East and West Ends. On this occasion, however, the two railway stations (W and L), one at either end of Princes Street, were correctly represented. One of the main streets leading off Princes Street was again reproduced on the same side as the Castle and the shops (SS), which in fact line the North side of Princes Street, were introduced on the South side. On a third attempt (fig. 4 (*d*)), drawn four months after admission, the patient began the task rather differently by orienting his plan vertically instead of horizontally on the paper. He was again uncertain as to the relative position of the East and West Ends, but eventually represented the Waverley Station (W) in its proper position. He was extremely doubtful as to which side the Gardens which line the South side of Princes Street lay and eventually introduced them on both sides of the street. This is a particularly gross error to anyone familiar with the city. The patient also displaced the Scott Monument (Sc) from one end of the street to the other and placed it on the wrong side. A prominent shop (J), in fact opposite the Monument, was correspondingly transposed in the plan.

We tested the patient's geographical knowledge in the wider sense by requesting him both to draw freehand maps of the British Isles and to complete maps drawn by ourselves. We also asked him to indicate on a blank map of the British Isles the positions of the principal towns, &c.

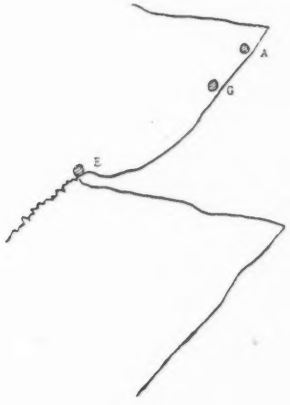

FIG. 5

Some of the results obtained on these tests may be briefly described:—

Map drawing.—A freehand *map of Scotland*, drawn four weeks after admission, is shown in fig 5 and is notable for the total omission of the left-hand side. Although Edinburgh was placed on the Moray Firth, Glasgow, which the patient admitted was to the West of Edinburgh, was introduced on the East coast not far from Aberdeen.

Orientation on maps.—When confronted with an ordinary blank map of the British Isles (excluding Ireland) Scale 150 miles to an inch, it was found that the patient could trace the entire outline with his forefinger when asked to do so and did not omit the left side. He was, however, quite unable to orient himself consistently in regard to it when asked to represent the position of some of the more important towns. Thus he placed Edinburgh on the Humber and, when asked to draw in the boundary between England and Wales, drew an oblique line from the Wash to the Severn and designated as Wales all that lay to the *right* of this

FIG. 6

line. Nevertheless, when requested he introduced London in its proper position. He correctly identified the Isle of Man on the *left* side of the map, but indicated Dungeness when asked to point out Land's End. Asked to draw in Ireland in its proper position, he represented it as lying off the Yorkshire coast. These features are shown in fig. 6. Rotation of the map through 180 degrees, undertaken under the patient's eyes and with his full knowledge, led to an even more severe disorientation. The patient continued to treat the map as though it were in fact upright and introduced London on the Solway Firth.

There was rather little improvement in the patient's capacity to orient himself with reference to a map during his stay in hospital.

(4) *Verbal Recall of Topography.*

The patient's capacity to describe routes formerly well known to him in Edinburgh was appreciably disturbed.

Topographical recall.—As a check on the drawing tests reported above, we often asked the patient to describe the order and sequence of the principal streets and buildings in various parts of Edinburgh with which he was well acquainted. On one occasion, for example, the patient was asked to detail the principal streets and buildings which he would pass in going along Princes Street from East to

West. A number of mistakes were made similar to those reported above on the drawing tests. Thus he mentioned that both the Castle and a street in fact opposite to it lay on the same side of Princes Street. He had also much difficulty in defining in terms of distance and direction the relative positions of outstanding buildings and landmarks in the city.

On re-examination after one year, it was thought that the patient's capacity to describe routes had considerably improved, although some mistakes were made and the patient might detail a roundabout route when asked to describe the shortest way from one part of the city to another. At this time, his memory in more general ways was entirely adequate and mistakes were no longer made in recalling past events in their proper chronological sequence.

IV. Discussion.

It would be idle to deny that this is a complicated case which presented, in the earlier stages at least, a number of features usually grouped in independent clinical syndromes. These comprise first, the amnesic syndrome with the exceptionally well-marked temporal disorientation; secondly, the left-sided agnostic syndrome of the type often associated with parieto-occipital lesions of the minor hemisphere; thirdly, the finer visual-constructive disabilities; and fourthly, the curious and outspoken disorder of topographical recognition, orientation and recall. So extensive a clinical picture has seldom been reported in cases of unilateral cerebral lesion, although it may be recalled that Hughlings Jackson (1876) described a case showing many of the features displayed by our present patient. This was a case of glioma in the right temporal lobe abutting on the floor of the posterior horn of the lateral ventricle. Jackson's patient experienced gross difficulties in route-finding and also made mistakes in dressing herself. There was also, apparently, some weakness of general memory and it is reported that the patient was liable to put sugar into the tea three or four times over. Although the patient was able on occasion to state correctly various (topographical) facts about her native town, mistakes were made and the patient appeared slow and perplexed in answering questions dealing with these matters. It thus seems fair to conclude that topographical memory was to some extent disturbed and the case thus anticipates those reported by Meyer (1900) to which reference has already been made. In Jackson's case, further, unlike those of Meyer, the localization of the lesion was verified at autopsy.

An interesting feature of Jackson's case, strikingly parallel to our own, is the disorder of *memory retention* to which special reference is made. It

is also worth pointing out in this connection that one, at least, of Meyer's cases showed at first the picture of an amnesic syndrome and that Spillane (1942) has more recently described a case of unilateral right-sided vascular lesion in which this syndrome was associated with left-sided anosognosia. The bearing of these findings upon the general problem of localization of the amnesic syndrome cannot be considered here but we cannot refrain from calling attention to a recent review by Delay (1942) in which a considerable body of evidence suggesting a focal localization for this syndrome is surveyed and evaluated (Delay, 1942, pp. 131-7).

The principal psychological problem raised by the present case is whether the topographical disorder is an independent syndrome or whether, on the other hand, it can be ascribed to deficiencies in other psychological fields, e.g. visual cognition or general memory. In so far as our patient's *general memory* is concerned, it will be borne in mind that the retrograde amnesia present on admission had largely cleared up at the time the major studies of spatial orientation were undertaken. It therefore appears unlikely that the disorientation can have been due to a retrograde amnesia of the type ordinarily seen in cases of Korsakoff's psychosis.[1] This conclusion is further borne out by the fact that we have more recently had occasion to observe defects of topographical memory closely akin to those shown in the present patient in a case of right-sided traumatic parietal lesion who at no time displayed an amnesic syndrome of the classical type. In this case, moreover, the disturbances of place-recognition and route-finding were markedly in evidence three years after the patient sustained his brain injury.

The possible influence upon topographic orientation and memory of defects in the general sphere of *visual space perception* must now be considered. The relevant defects in the case under discussion comprised a very marked agnosia for the left half of visual space and a notable failure to organize and manipulate high-grade spatial relationships in the central field of vision. These symptoms were further associated with the characteristically restricted and incoherent mode of visual exploration which

[1]We may comment that topographical memory appears to have been little studied in the many cases of amnesic syndrome of which detailed psychological reports are available (e.g. Liepmann, 1910; Stoerring, 1936). In one case of chronic alcoholic Korsakoff psychosis that came under our personal observation we gained the impression that memory in its topographical aspects was intact despite extensive retrograde amnesia for events. On the other hand, some deterioration of topographical memory has been reported in a case said to have presented a pure amnesic syndrome (Chaslin and Portocalis, 1908). We need hardly point out that detailed tests of orientation in home surroundings are seldom practicable in cases of this kind. Further research on the problem is obviously needed.

we have termed elsewhere the "piecemeal approach" (cf. Head, 1926; Paterson and Zangwill, 1944*b*). It is prima facie likely that all these factors were involved to some extent in the patient's orientation disabilities. In particular, the constant tendency to veer or turn to the right rendered it virtually impossible for the patient to find his way about no matter how well he was acquainted in the past with the particular spatial setting. The left-sided agnosia, which obviously underlay this phenomenon, was also clearly responsible for many of the omissions and errors in plan-drawing and map-reading and very probably interfered to some extent with the patient's capacity to recognize and recall familiar routes and buildings. The visual-spatial disorder, moreover, as conceived in terms of the tendency to respond to individual details without reference to their wider spatial context, undoubtedly added further to the orientation disorder and the difficulties in route-finding. Indeed the patient commonly gave the impression that he had lost the capacity *visually to survey an extended spatial situation.* This would obviously limit the extent to which available orienting cues could fulfil their normal function in conditioning recognition. We may therefore conclude that the unusually gross disorders of visual perception and exploration contributed in no small measure to the outstanding disturbance of orientation.

It must not, however, be forgotten that the case presents certain features suggesting that the causes of the topographical derangement go beyond visual perception in the narrower sense of the term. In the first place, it will be remembered that many of the cases with spatial-agnostic disorders (with or without neglect of the left half of space) reported in the literature did not show comparable disturbances of orientation and spatial memory (Poetzl, 1928; Scheller and Seidemann, 1932; Russell Brain, 1941). And in the second place, a number of our patient's errors of place-recognition suggested a specific derangement of those cerebral mechanisms or schemata, built up through past experience, which must be assumed to underly the recognition of topographical data and thereby mediate orientation. Thus the patient's notable failure to identify Arthur's Seat (an unmistakable hill and landmark in Edinburgh) is clearly a case in point. The patient was allowed to survey the hill from a distance sufficiently great to allow perception of its whole outline without shift of ocular fixation and the fact that he recognized it as a hill (which he misidentified) is clear proof that it was actually perceived. It might, of course, be argued that the misidentification was conditioned by the patient's uncertainty as to his immediate whereabouts, but even this does not account for the fact that perception of the hill failed to re-orient him

securely (as it certainly would in the case of a normal person uncertain as to his precise location). In any case, we were able to observe and record many further cases of recognition failure (e.g. the patient's failure to recognize his church) under conditions in which he was in no doubt as to his immediate whereabouts and could name the street in which he was situated. The same applies to the patient's inability to identify his own house which was witnessed on at least two separate occasions. In such cases, we are obliged to conclude that despite adequate orientation and perception the patient was unable to bring the perceived material into proper relation with those experience-schemes or memory-dispositions which normally render place-recognition immediate, automatic and correct. This failure, in sharp contrast to the patient's invariably accurate recognition (gnosis) of objects, colours and written symbols, appears to warrant treatment as a *specific topographical agnosia.*

A proper assessment of the associated disorders of *topographical representation and recall* is hard to undertake. As regards the errors in plan-drawing, we may recall that very similar findings have been obtained in cases of unilateral lesion showing related types of visual-spatial agnosia (Head, 1926; Scheller and Seidemann, 1932; Paterson and Zangwill, 1944*b*). It is therefore entirely possible that the more immediate disorders of perception would adequately account for them without the necessity for assuming an additional impairment of topographical memory proper. At the same time, the patient's inability to *formulate* correct topographical relationships, despite the absence of aphasia, would rather suggest an additional disorder in the field of spatial memory. This might quite possibly depend on a defect of visualization of the type long ago described by Wilbrand (1887). We may, in any case, conclude that ideational representation suffers in much the same way as topographical perception and is a function of a specific inability to maintain a coherent scheme or frame of reference in the visual sphere.[1]

A brief comment is in place on the patient's outspoken tendency to make use of *symbolic cues* (tram-car numbers and the like) to assist him in spatial orientation. This feature is not only in itself interesting but strikingly reminiscent of the orienting behaviour shown by Meyer's cases and may represent a variety of compensation occurring spontaneously.

[1]A more elaborate theory along these lines has been worked out by Poetzl (1928) and has been considered briefly by Russell Brain (1941). We hope to consider the theory of spatial disorganization in greater detail in subsequent communications. For the present, we shall comment merely that our present case appears to provide some confirmation of Poetzl's general views.

At a later stage, further, we noted some tendency for the patient to make use of *outstanding non-symbolic perceptual detail* as orienting data. An example of this was his deduction as to his whereabouts from the prominent red colour of a neighbouring building. Cues of this kind are, of course, regularly employed by normal persons in orienting themselves in unaccustomed surroundings but soon drop out as orientation becomes habitual. It was interesting that the patient displayed some insight into this tendency in the course of recovery and was able to exploit it intentionally in the interests of proper orientation.

It may be asked in conclusion why the topographical memory loss in the present case (and equally in those of Jackson and Meyer) was relatively so much more pronounced than in the otherwise comparable cases of right-sided cerebral lesion reported by Scheller and Seidemann (1923), Russell Brain (1941) and others. One possible answer to this question, which has already been given by Nielsen (1937, 1938, 1940), is to be found in the concept of occipital dominance. According to Nielsen, visual object-agnosia along with topographical memory loss is produced by lesions of the dominant occipital lobe—generally the left, but in some cases the right, even in right-handed persons. It might therefore be assumed that in the present case the right cerebral hemisphere was dominant in relation to visual-associative functions. Such an argument does not, however, explain the absence of visual object-agnosia, and it will be remembered that it is on this symptom that Nielsen (1937) places greatest weight as the criterion of occipital dominance. Further, there are other and more general reasons for accepting Nielsen's conception with reserve.

A second possible answer might be that the lesion responsible for the topographic memory loss in the cases under discussion was in fact bilateral, and we know from the work of Holmes (1918,1919) that this symptom is regularly found in those cases of bilateral parietal lesion who show visual disorientation. In our present case, this explanation cannot, of course, be absolutely excluded but it appears to us improbable.

A third possible answer, and one to which we ourselves incline, is that the nature and location of the lesion in cases of this kind are directly responsible for the topographical memory loss. What appears to us the characteristic lesion in the present case is the gross loss of brain tissue around the lateral ventricle in the region of the vestibule and temporal horn. The loss is not confined to the lateral surface but affects the medial side also. It is, of course, clearly impossible to argue generally from the findings in a single case and many more detailed cases of visual disorientation both with and without strict topographical memory loss will be

needed along with autopsy results before we are in the position to ascribe definite localizing evidence to this symptom. We may, however, point out that some support for our argument is to be found in the post-mortem findings in Hughlings Jackson's case to which reference has already been made. In this case, a tumour in the right temporal lobe was found which abutted on the floors of the posterior and temporal horns of the lateral ventricle and appeared on the inferior medial surface of the hemisphere at the level of the lower end of the parieto-occipital fissure. The area invaded by the tumour corresponds rather closely with the area of tissue destruction in our present case.

Lastly, it will be borne in mind that although the lesion in both Jackson's case and our own was right-sided, there is evidence that the syndrome of topographical memory loss may also be associated with comparable unilateral lesions of the left cerebral hemisphere. Indeed on Nielsen's view, as we have seen, this is the more usual expectation. We have ourselves recently had occasion to study a case of left parieto-temporal traumatic lesion which presented, in addition to gross jargon aphasia, pronounced disorders of spatial orientation and memory. This case will be communicated in the near future. It is true that a relatively large proportion of the cases of topographical disorientation reported in the literature have been cases of right-sided lesion, but this may be due neither to occipital dominance nor to some special role of the right cerebral hemisphere in spatial orientation as has been postulated by Lange (1936). It appears to us plausible to suppose that topographical memory loss may be produced by an appropriate unilateral lesion of either hemisphere and in this respect our conclusion is identical with that to which we were earlier led by a study of related types of spatial-agnostic defect (Paterson and Zangwill, 1944*b*). In the cases with left-sided lesions, however, the presence of aphasia and kindred defects greatly impedes careful study of visual perception and may effectively mask defects in the general sphere of topographical memory. At the same time, we believe that further work on the respective contributions of the two hemispheres to the organization of topographical memory will be needed before valid general conclusions can be safely drawn.

V. Summary.

(1) A case of traumatic right-sided focal cerebral lesion is described in which disorders of topographical orientation and memory were exceptionally pronounced. These disorders have been studied in detail and the findings reported in full.

(2) The relation of the topographical defects to concomitant disturbances of visual perception, general memory and the body scheme is considered. It is concluded that the patient sustained, in addition to these disturbances, a specific topographical memory deficit or agnosia which outlasted the cognitive disabilities in other fields.

(3) The bearing of the present case upon problems of cerebral localization and its significance for general psychological theory are briefly discussed.

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LEGENDS FOR FIGURES.

FIG. 1.—X-rays of skull showing location of missile (*a*) Posterior view and (*b*) Lateral view.

FIG. 2.—Air-encephalograms: (*a*) Posterior view; (*b*) Lateral view.

FIG. 3.—Ground-plan of house: (*a*) Drawing by patient's wife; (*b*) Drawing by patient.

FIG. 4.—Plan-drawing: (*a*) Plan of Princes' Street, Edinburgh; (*b*), (*c*), (*d*) Plans drawn by patient

FIG. 5.—Map of Scotland drawn by patient.

FIG. 6.—Completion of a map by patient.

FOLIE À DEUX IN UNIOVULAR TWINS REARED APART.

BY W. H. CRAIKE AND ELIOT SLATER [1]

WITH THE ASSISTANCE OF
GEORGE BURDEN.

INTRODUCTION.

UNIOVULAR twins who have been separated in the earliest years of infancy and have been brought up apart from one another are phenomena of great scientific interest. Owing to their uniovularity, both members of such a pair have identical hereditary equipment, and any differences between them must be attributed to environmental influences. Most twins are brought up in the same home, and thereby come under the same sort of education and training, and under very similar influences from parents, brothers and sisters and comrades. They are therefore likely to be more alike one another than hereditary factors alone would ensure. Furthermore, the psychological relationship of one uniovular twin to the other is of a peculiarly intimate and, as a rule, affectionate kind; so that when they are brought up together they are likely each to influence the development of the other. The mutual relationship, and the similarities of environmental influence, do not hold in cases where the twins have been separated in early years, and we then see a controlled experiment of nature in an almost pure form. Unfortunately, uniovular twins who have been separated in infancy are exceedingly rare. After intensive search extending over many years, Newman, Freeman and Holzinger were only able to discover in the whole of the U.S.A. nineteen such pairs, which they have reported at length (1937). None of their pairs had any mental illness and, as far as we know, the case here reported is unique in the literature.

The twins here described were separated at the age of 9 months, were brought up in different families in different parts of the country, met each other for the first time at the age of 24, never lived together or had any close contact with one another, but each in due course developed a paranoid psychosis, each involving the other in her own system of delusional ideas. It is this mutual involvement in a paranoid

[1] With the aid of an expenses grant from the Medical Research Council.

system which suggested the title of "folie à deux," although the possibility that the psychosis of either "induced" that of the other has, we think, to be rejected.

The first member of the pair to appear was Florence. She was referred to the Maudsley Hospital Out-Patient Department from the Observation Ward of Fulham Hospital by Dr. Alexander Walk, to whom our thanks are due. She was there examined by both of us, and her twin was eventually contacted and examined in her own home (by W. H. C.). Attempts were made to get information from external sources about the twins. All the relatives are dead, and the Children's Home, where Edith was brought up, was closed years ago. Contact was, however, made with Edith's employers, as will later appear.

History.

The father of the twins was a farmer in South Wales, but after the death of the mother he sold the farm and became a coal-miner, and later married again. He was at first an irreligious man, but was converted by his second wife. They used to "live in Church" on Sundays, and he became a Sunday-School teacher. Edith says he was a very violent man and drank heavily. She lived with him until she was 8½, and was terrified of him. There were always family scenes, and she still bears on her head a scar where he cut her with a razor. Of the mother nothing is known except that she died about the age of 35, nine months after the birth of the twins. There was a boy born previously, who was about 5 at the birth of the twins, but he is said to have run away from home and to have died in 1914, when he would have been 27.

Immediately after the death of the mother, Florence was adopted by an unmarried maternal aunt and went to live in her home. Edith stayed on with her father, to lead a miserable childhood. She walked in her sleep, as a child, and before her first confession, which she dreaded, had nightmares of being flogged. About the age of 8½, her father put her into a Children's Home, where she lived until she was 19. She did quite well at school, and says she reached the top standard (VII) two years before leaving school at 15½. She then started working on a farm, but developed "nervous dyspepsia and anæmia," and was in Cardiff Workhouse at 20. Her father took her back into his home from the workhouse, but soon told her to get out, and she ran away to live with an uncle. During all this time she had known of the existence of her twin, but had never met her or corresponded with her. She says, however, that even when she was living with her father, Florence was making trouble for

her by writing to her father and telling him Edith had told her that he was a drunkard. From Wales Edith came to London to seek employment. Her uncle paid her fare, but Edith says Florence claims that she paid it and that Edith is under an obligation to her. Edith first met her sister when they were 24. A little money had been left them by an aunt, and the solicitor got in touch with both of them. Even at this first meeting Florence accused her of stealing a ten-shilling note from her purse, quite untruly; the only money Edith ever stole was from her father when she was living with him. The sisters saw a little of one another, but could not get on. Florence used to spy on her and write anonymous letters to her employers. About this time Edith saw Florence in the street on a bicycle with a man; she recognized her, because she was wearing a yellow velour hat, and knew she was spying on her.

Edith continued to live in London, working as a domestic. She has excellent references from her employers, and seems to have been of quiet, reserved disposition, with few friends, but hard-working and of stable mood. She has always been keenly religious and an Anglo-Catholic. Her last employer, who kept her for eleven years until 1940, reports that she had to get rid of her in the end because of her delusionary ideas, "which led her to believe that not only her own friends and relations but practically everyone who visited the house accused her of wrong doing. Apart from these ideas she was an excellent woman in the house, perfectly honest, willing, trustworthy and possessed of a kindly disposition. . . . The two sisters did not see much of each other and when they did meet they could never agree. Edith always gave me to understand that when Florence did call to see her it was to seek financial help." After this, Edith was directed to a glass factory. The Welfare Officer there says that she is very good at her work, and though the firm is cutting down staff, she would be kept as long as she wanted to stay. She seems quite happy there, but has lately suggested that she would like her release to become a nun. The only thing odd about her is that from time to time she asks to see him and inquires whether people are talking about her, and whether she is causing any trouble.

During these latter years, Edith has continued to have reason to complain of Florence's behaviour. As recently as November, 1944, she has seen Florence watching her house. She thinks she must be jealous. She has had a very hard struggle, everybody has been against her, and she has lost many good jobs in domestic service. All this is entirely due to the influence of Florence.

Florence was brought up by her mother's sister, and made her home

with her until her death in 1944 at 85. She was very happy with her. Her childhood was happy, though she was rather nervous and frightened of the dark; she did not walk in her sleep and remembers no nightmares. She attended an ordinary elementary school where she was rather backward, leaving at 14 from the third standard from the top (IV). This was partly due to missing a lot of schooling because of fainting attacks. She says she has picked up a lot afterwards from reading. After school she got a post as a children's nursemaid. She took some of her employer's soap, and was charged with the theft and put into a convent for two years. While there she was happy. Ever since then she has worked as a domestic, and has risen as high as head housemaid. She has had regular employment, and in intervals between jobs has stayed with her aunt. About the age of 18 she had an attack of "nervous debility," with symptoms of abdominal pain, vomiting and nerviness. She went to stay with another aunt in Wales and recovered. Apart from that, she has had no nervous trouble in the past. She describes herself as of quiet but cheerful disposition, always of much the same mood, not inclined to be suspicious, and with many friends. She was very religious in her youth, especially at the time of her confirmation at 16; she kept up going to Communion for many years, but has been less regular and interested "since I've been like this." In 1916 her aunt died, from whom the money came, and in 1917 the money was divided between her and Edith, whom she then met for the first time. She noticed at once how like they were, and rather took a fancy to her. She met her sister twice more, and then the letters began. Out of the blue, Edith wrote her several letters accusing her of spying. About that time a fellow servant at her place of employment, who had been reproved by Florence, said to her, "You think a lot of yourself, but you've been to prison." Florence thinks that it must have been through Edith that knowledge of her being charged for theft in the past had got around. It was about that time that Edith wrote to her, "Why do you come down on the back of Alf's bike spying on me? I know it's you because of your lemon hat and brown coat." There was no more trouble until years later. About three-and-half years ago, "people said they knew all about me. They (the staff) said, 'Why don't you get married? That's what's the matter with you.'" "There was a general undercurrent." "It was after I knew her that all this started, and I began to wonder whether it was her. She must have been spreading rumours about me. But you can't accuse people unless you're absolutely sure." About a year ago Florence met Edith again. "We spent one or two nice week-ends. I mentioned I had a chance of two rooms and should we take them. She said no, she wanted

me to keep away. She accused me that Sunday of going to neighbours saying she was mental."

On 27.12.44 Florence was admitted to the Observation Ward. The notes there read:—

"Admitted. Physically n.a.d Mentally very depressed, weeps and sobs copiously as she tells of all the undercurrent against her. People say she is mad, she is mental, they throw up at her all her past life. She is determined to get at the bottom of it. Only once did she actually catch a voice, a man's voice, saying horrible things. 29.12.44: Seen by magistrate. Continues to complain of some undercurrent going on against her. 6.1.45: Improved, still some depression but not so worried by delusional ideas. Seen by Dr. Walk who notes: depressed and preoccupied, although she says she feels much happier under care. Only one remark has been passed about her since admission, but there were plenty before. . . . She says that among other things the voices have told her that she is mental. they have accused her of having been in prison."

Florence continued to improve, and shortly after having been sent for examination to the Maudsley Hospital she was sufficiently well to be allowed home.

Findings on Examination.

Physically.—The twins had to be examined separately, as they could not be induced to meet. Edith was interviewed in her own home, Florence at the Maudsley. Physically, there were no abnormal findings other than a slight degree of deafness, probably due to otosclerosis. Florence stated that she began to go deaf about three years ago, and had been examined at the West London Hospital. Inquiries at that hospital showed that no one of her name had attended in the E.N.T. Department during that time. As Edith refused under any circumstances to have her ears examined, it was not thought worth while pursuing the matter. Florence states that her twin is not the least deaf, and that Edith has accused her of not being deaf herself but pretending it. Actually both twins are of about the same degree of deafness, needing no instrument but hearing well with some raising of the voice.

Both twins are of middle height, and of dumpy pyknic build, with broad face and slightly cleft chin, broad-based retroussé nose, and a suggestion of widow's peak in the hair-line over a high rounded forehead. In both hair is abundant, a dark brown with streaks of grey, eyes grey with some brownish flecks. The voices are extremely alike, and listening to either one might imagine the other was speaking.

Comparison of the finger-prints shows the following pattern types and ridge counts (count to radial triradius given first; W, whorl; DL, double loop; UL, ulnar loop):

Florence:	R. 1. W 18, 25	2. UL 15	3. UL 18	4. UL 17	5. UL 19
	L. 1. DL 13, 21	2. UL 14	3. UL 16	4. UL 17	5. UL 15
Edith:	R. 1. W 18, 24	2. W 14, 13	3. UL 16	4. UL 13	5. UL 17
	L. 1. W 16, 20	2. UL 13	3. UL 14	4. W 20, 6	5. UL 15.

These prints show a closer similarity between the twins than between their right and left sides. The Florence-Edith product-moment correlation coefficient is + 0·84, the right left correlation coefficient is + 0·82.

General appearance and bodily constitution are in favour of uniovularity; there is a striking facial resemblance between the twins, in addition to general con-

formity of build and colouring. There are no points of physical discrepancy. The degree of similarity of the finger-prints is practically conclusive of uniovularity.

Mentally.—When seen at the Maudsley Hospital on 8.1.45, Florence still had feelings of an undercurrent against her, and said she had heard opprobrious remarks being passed, but was unwilling to go into detail. She thought the undercurrent was Edith's work; Edith had been accusing her for many years. She was slightly suspicious and reserved, but there was no marked degree of depression and affective rapport was good. There was no evidence of any shallowness of affect or of any bizarre quality, nor of any schizophrenic features in attitude, expression or motor behaviour. She did not appear to be hallucinated. Intellectually she was well preserved. She was seen for a second time on 29.1.45. She was then back at work and feeling very well, which she attributed to taking a course of "sanatogen." She said she had neither seen nor heard further sign of the "undercurrent." She was seen for a third time on 16.7.45, and then showed no abnormal sign at all. She was working at a hospital as a domestic, but did not like it and did not feel she could go on doing it, although she got on all right with the others. She had met Edith the week before Easter, and remarked that her sister seemed nervous of her.

Edith was examined in January, 1945, at home. She was found to be living in one room in an old Victorian house converted into flatlets. She gave the history in quite co-operative manner, but during the interview became very emotional and tearful. She was seen again on 18.2.45, when the finger-prints were taken. She was very suspicious of these proceedings. She said that when she first came to her flat the other boarders in the house avoided her; they had since become more friendly, but now they avoided her again, because she is no good. One of the men in the house had told her to get away, because the police were after her. The police are checking up on how much she earns and spends, although she has done no wrong. The police car stops outside her house every night, and they are watching her. Last year someone in the house next door committed suicide, and that house and the one in which she lives were full of police, and there was a policeman stationed outside. They were watching her. Her sister may be connected with all this, because she has told Edith's employers that Edith is immoral and has an illegitimate child, which is not true. Edith was seen for a third time on 16.6.45. She said she had seen Florence in March, meeting her by chance in a Boots in Ealing on the way to Church. At first she did not recognize her, she looked so well. Florence was very annoyed with her, and accused her of having said she was mad; this, Florence said, was not true, because she had been discharged from hospital on 12.1.45 and had not returned again. The whole of this was news to Edith. The two sisters went to Church together, but Florence took no part in the Service, did not speak again, and "took to her heels" on leaving. Edith does not know what she was doing in Ealing, and presumes she was spying again. The general undercurrent against Edith continues; someone is prying into her affairs, and she suspects it is Florence. She has never seen any of the letters about her, but has learned of them in roundabout ways. She recently went to St. Bernard's Hospital to apply for a job, but declined it because she was upset by the patients she saw. Now people are saying she has been a patient in the Asylum. Edith also complains she has been having "very severe turns" in which she falls like a log. They began on Easter Monday, are becoming more frequent, and are leading to absence from work. This has aroused a lot of comment. In these later interviews Edith was inclined to be suspicious and reserved, but there were no characteristic schizophrenic symptoms. Intellectually she was well preserved, but probably of low average intelligence.

Comment.

These uniovular twins were brought up along entirely different lines. Florence had a stable home with an affectionate maternal aunt, and has lived with her all her life until a year ago. Edith lived with a drunken and violent father of whom she was terrified, and was put into an institution from 8½ till 19, and since 20 has lived on her own. Nevertheless their careers bear very striking resemblances. Both were nervous as children: Edith walked in her sleep and had nightmares, Florence had fainting attacks and was nervous of the dark. Both stole as children or adolescents, Edith from her father, Florence from her employer; in the paranoid state which each subsequently developed, stealing enters into the delusional content. The school careers are said to be very different, Edith reaching the top standard, Florence only the fourth. But the intelligences of the twins when adult are probably about the same, and the different success at school is probably due to adventitious circumstances. Edith at the age of 19 and Florence at 18 had some form of gastric disturbance, accompanied by indications of a neurotic aspect, which in each case passed off rapidly. Both went into domestic service. Neither married, though Florence says Edith was once engaged. A sexual element enters later into the delusions of both: Edith thinks she is accused of having an illegitimate child, Florence says people have hinted she is sex-starved. Their personalities are very similar, although on the whole Florence is the more open, sociable and conciliatory of the two; both are religious, Edith the more so. Both have got gradually deaf, and both imply that the other merely pretends it. Paranoid symptoms appear in each about the same time, soon after their first meeting. In the case of Edith, there is a suggestion of a still earlier onset, but this may be due to a falsification of memory. From the first Edith directs her suspicions against her sister, Florence however against her fellow employees. Apparently Florence does not begin to believe evil of Edith until the latter's accusations have begun. It seems improbable that the onset of the psychosis was in either case associated with deafness; the deafness even now is slight in both, and it can hardly have existed to an effective extent in the twenties. The development of the paranoid state was in Edith quiet and insidiously progressive, with good preservation of personality and without social decline. With Florence, too, the personality remains well preserved, and symptoms appear only episodically at times of stress, and eventually more floridly in the involutional period. At the height of this episode, which is quite short-lived, appear auditory hallucinations and a severe depressive reaction, which are never shown by Edith. On the whole, however, Edith's

state is the graver one. It is tempting to relate the different degrees of severity to the more fortunate upbringing and early life enjoyed by Florence. The psychotic symptoms, as already remarked, show many resemblances, even in the content of the delusions. It is rather remarkable that, despite the religious interests of the twins, neither shows a religious colouring to the mental content.

In the opinion of the authors, the diagnosis in each case must be one of an endogenously determined chronic paranoid state, probably basically schizophrenic in nature. The illnesses at 18 and 19 may in each case have been mild schizophrenic attacks, though there is no positive evidence to support this. The mild degree of deafness, from which both twins suffered, probably did scarcely more than favour the paranoid development. The possibility that Florence's illness was induced by that of Edith, and is psychogenic in nature, and that we have a case of *folie à deux* in the classical sense, must be rejected. Florence's acute episode was in no way related to any action on Edith's part, and the other occasions on which she became more openly paranoid are equally unrelated to Edith's behaviour. Edith's report of recent falling attacks remains an unexplained feature. A late symptomatic epilepsy cannot be excluded, but equally cannot be confirmed, as nothing will bring Edith to go to hospital. The possibility of a constitutional tendency to manifestations of the epileptic type is also suggested by Florence's fainting attacks in childhood.

The development of the inter-twin relationship is unusual and interesting. Unlike the extremely affectionate bond which usually unites uniovular twins, the relationship here is one of suspicion and dislike. Florence says that when they first met, she took a fancy to her sister, and hints that more than once since then she has made gestures of reconciliation; her attitude throughout is much the less unfriendly of the two. Edith, however, was hostile from the first. It seems possible that this was caused in the first place by resentment at her own less fortunate lot; but that the attitude once set up caused its own perpetuation and aggravation. Each twin occupies for the other an over-valued position; to each the other is supremely important, although the circumstances of their lives touch at few points. Edith at first sight places Florence in the centre of her persecutors; Florence, with her own inborn tendency to paranoia, reacts to this by coming in her turn to regard Edith as her chief enemy.

SUMMARY.

Two uniovular twin sisters of 52 are described, who were separated at the age of 9 months, but each in due course developed a paranoid

psychosis. Concordance of childhood neurosis, of life story, of personality, and of eventual symptomatology are shown, despite very different upbringings. The principal difference between the psychoses is that while one shows a chronic insidiously progressive paranoia, the other shows paranoid symptoms only episodically. Each sister centres her delusions around the other; this appears in one of them to be reactive to the accusations of the other. Both are regarded as being the subjects of a basically schizophrenic illness.

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THE DEVELOPMENT OF THE HUMAN LATERAL GENICULATE BODY.

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THERE is a large amount of literature relating to the lateral geniculate body and its association with the thalamus. Attention has been drawn to the phylogeny of this body, and in this connection should be mentioned the work of Rioch (1929-30), Papez (1929), Kuhlenbeck (1935-36), Gurdjian (1927), Ingram, Ranson and Hannett (1932), Tsai (1925), Kappers, Huber and Crosby (1936), Winkler and Potter (1914), Herrick (1917), Durward (1930), de Lange (1913), Bellonci (1888), Munzer and Wiener (1898), Edinger and Wallenberg (1899), Woollard (1926), Woollard and Beattie (1926-27), and Le Gros Clark (1932*a*, *b*, *c*; 1940-41).

The general agreement with regard to mammals is that the lateral geniculate body consists of two parts or nuclei, dorsal and ventral, first identified by Kolliker. Clark (1932*b*) believes that the ventral nucleus of the mammalian lateral geniculate body has its homologue in the lateral geniculate body of reptiles. From his own comparative studies this author finds that the ventral nucleus in primitive mammalian brains is a "differentiated lateral segment of that part of the pars ventralis diencephali which is called the subthalamus" (1932*b*). He puts forward in support of this contention two observations: first the topographical relations between the ventral geniculate nucleus and the subthalamus, and second the merging insensibly of the ventral nucleus into the zona incerta of the subthalamus with identical cells in the two. In higher mammals Clark (1932*b*) found that in the developing thalamus the continuity of the ventral geniculate nucleus with the subthalamus is very evident: but in the adult brains the association of the ventral geniculate nucleus with the zona incerta is not so obvious by reason of the lateral displacement of the former which is partly due to the greater development of the lateral and ventral thalamic nuclei. From its fibre connections Clark regards the ventral nucleus as "essentially a subthalamic optic centre with tectal connections" (1932*b*).

In higher mammals the ventral geniculate nucleus becomes progressively smaller as the telencephalon overwhelms the subthalamic centres. In the higher primates Clark (1932*b*) recognizes the ventral nucleus as scattered cells among the fibres of the optic tract as these proceed

towards the main dorsal nucleus of the lateral geniculate body. These scattered cells have been named the pregeniculate nucleus by Woollard (1926). In the human fœtus of 9·2 cm. (14 weeks), Clark found that the ventral nucleus is as distinct as it is in simple mammalian brains (1932*b*).

As regards the dorsal nucleus of the lateral geniculate body, Clark states "comparative and embryological data indicate that this dorsal nucleus is to be regarded primarily as a differentiated lateral part of the ventral nuclear mass of the thalamus" (1932*b*). In primitive mammalian brains he asserts that the dorsal geniculate nucleus consists of the same type of cell as the ventral thalamic nucleus. Furthermore he describes the dorsal geniculate nucleus as being applied to the lateral aspect of the ventral thalamic nucleus, the lateral medullary lamina intervening between the two nuclei. This topographical relation Clark also mentions in a reference to the thalamus of a fœtus of 9·2 cm. (14 weeks) (1932*b*), which he refers to as an "early human embryo."

On the other hand Goldby (1940-41) in his paper on the thalamus of phalanger states that the lateral nucleus of the thalamus is demarcated into three divisions, A, B and posterior. The posterior division of the lateral thalamic nucleus (i.e. pulvinar) is further subdivided into medial and lateral parts. According to this author the "lateral part of the nucleus lateralis posterior (pulvinar) cannot be distinguished from the medial part of the lateral geniculate body in preparations stained to show cells." Both contain medium and small cells of the same type. In Weigert preparations, however, Goldby distinguishes between the two on account of the dense network of fibres within the lateral geniculate body which is not seen in the pars posterior of the lateral thalamic nucleus.

In her description of the early development of the human diencephalon, Gilbert (1935) states that the lateral part of the dorsal thalamus (i.e. thalamic portion of the diencephalon) gives rise to the pars dorsalis of the lateral geniculate body, while the ventral thalamus (i.e. subthalamic portion of the diencephalon) gives rise to the pars ventralis of the lateral geniculate body. According to Kappers, Huber and Crosby (1936) the lateral geniculate body belongs essentially to the lateral division of the dorsal thalamus.

As is well known the dorsal geniculate nucleus in higher mammals is connected by fibre pathways to the area striata of the occipital cortex. The dorsal nucleus exhibits lamination. Primarily, according to Clark (1932*b*), this is trilaminar, but subsequently six layers are defined in association with the appearance of a hemi-decussation at the chiasma.

The purpose of this paper is to trace the development of the two nuclei,

dorsal and ventral, of the human lateral geniculate body and to describe the onset and establishment of the lamination. The development of the nuclei in the human diencephalon has not received the same attention as other anatomical aspects. The embryological works of His (1904) and Streeter (1912) barely touched on the nuclear differentiation. Kuhlenbeck (1935-36) described certain nuclei in a few human embryos only. Gilbert (1935) recently studied the ontogeny of the human diencephalon, and reference to her findings will be made from time to time.

In the present investigation the external appearances of the diencephalon and brain-stem were noted at different stages of development, except in the small embryos which were sectioned in their entirety. In fœtuses up to 5·0 cm. length (10 weeks) the caudal extremity of the thalamus meets the tectum of the mid-brain dorsally and a shallow depression marks the union. The thalamus shows no projecting portion posteriorly. At 6·3 cm. (11½ weeks) a small postero-lateral prominence is seen at the caudal extremity of the thalamus which slightly overhangs the lateral aspect of the tectal region of the mid-brain. The latter itself exhibits at its cephalic extremity a lateral bulging (fig. 1). On tracing the optic tract it is evident that the postero-lateral prominence of the thalamus is associated with the tract. A specimen of 7·0 cm. presents similar features, but the dorso-lateral thalamic prominence is relatively more pronounced (fig. 1).

At 8·2 cm. this prominence is displaced from its contact with the quadrigeminal lamina (tectum of the mid-brain) by a second small elevation. This appears in the same transverse plane as the cephalic part of the collicular mass (fig. 1). Specimens of 9·0 and 9·2 cm. are similar but slightly larger. The optic tract can be followed to the original more rostral elevation. The inferior brachium, extending from the inferior colliculus, is seen to widen out proximally and to join the second elevation. It will be surmised that the rostral prominence is the lateral geniculate body and the more caudal one the medial geniculate body.

In the following account, the descriptions apply to serial sections all cut in the same plane, i.e. in the plane of the optic tract as it curves dorsally round the crus. In the case of embryos below 30 mm. length, serial sections of the whole brain *in situ* were made, beginning at the most cephalic extremity of the embryo. Thus the sections represent a dorso-ventral view in line with, or parallel to, the optic tract.

In the human embryo of 8·5 mm. (4½ weeks) the diencephalon appears as a somewhat oval tube from the anterior and ventral part of which extend the optic outgrowths (fig 2). Just behind these the minute hypophysial evagination is seen in the floor of the tube. From the lateral

walls of the diencephalic tube the budding of the cerebral vesicles has commenced. The optic stalks are hollow and communicate with the diencephalic cavity, and the optic nerves are not yet formed. The wall of the diencephalon consists of ependymal, mantle and marginal layers, and is slightly thickened by cell proliferation at its lateral aspect.

Already a modification of the shape of the diencephalic cavity is seen at 13 mm. (5½ weeks), created by a bulge towards the lumen on each side consequent on the greater enlargement of the dorsal part. In this way the dorsal (thalamic) and ventral (hypo- and subthalamic) portions of the diencephalon are demarcated, and the hypothalamic sulcus is defined between them on the lumen aspect.

The mantle layer has increased its cellular content in both the thalamic and hypothalamic parts. In the latter a distinct collection of cells is apparent, indicative of initial differentiation in the hypothalamus prior to any in the thalamus. Gilbert describes similar features at 12 mm., and refers to the first differentiation in the hypothalamus as the hypothalamic cord (1935).

By 16 mm. (6 weeks), the rostral part of the diencephalic cavity has a distinct hypothalamic sulcus which distinguishes the thalamus dorsally from the hypothalamus and subthalamus ventrally. In the latter the convex lens-shaped subthalamic nucleus is now defined, thus marking the subthalamus as the lateral part of the ventral region of the diencephalon, and the true hypothalamus as the medial part adjoining the cavity (fig. 3). Gilbert recognized the subthalamic nucleus in the 18 mm. embryo (1935).

In the more caudal part of the diencephalon at this stage (16 mm.), the thalamic mass forms the dorsal two-thirds, while the subthalamus forms the ventral one-third. This means that the thalamic mass has outgrown the subthalamic region. Moreover the dorsal thalamic region itself is now seen to be separated by a relatively cell-free zone into a dorsal and a ventral part, the latter being wider (fig. 3).

At the rostral extremity of the diencephalon at this period the optic stalks are still hollow and the central (hyaloid) artery is seen within the fœtal fissure (Mann). Retinal neuroblasts emit processes which gather towards the point at which the artery enters the optic cup. The processes, however, do not extend beyond this point and the optic stalk is still hollow.

In the embryo of 19 mm. (6½ weeks), the optic nerve is fully formed by fibres issuing from the retina which fill up the hollow optic stalk. The nerve appears as an elongated strand which is attached to the optic recess of the diencephalon at the ventro-lateral angle. The nerve fibres enter the marginal zone at the lateral angle of the optic recess, and are directed

medially for a short distance toward the mid-line. Here they are lost to view, and there is as yet no distinct chiasma except in so far as transverse fibres occur in the marginal zone; but such fibres are visible in a corresponding position in the mid-brain, and are regarded as simple commissural fibres. According to Frazer (1940) the optic stalk is filled during the sixth week by fibres originating in the retinal cells. This is confirmed by the present series.

By the 22 mm. stage (7 weeks), the chiasma has been completed and the optic tract can be traced from it over the lateral aspect of the diencephalon in a dorsal direction. Here it approaches and enters a peripheral collection of cells which has spread from the thalamic mantle layer. The spread of cells has occurred particularly in the dorsal part of the thalamus, and is related to the optic tract which reaches this region. The mass of proliferation in the mantle layer of the thalamus is limited, and a cell-free zone separates it from the ventral portion of the thalamus which in its turn approximates to the hypothalamus at the level of the hypothalamic sulcus. It would seem therefore that from the outset the optic tract applies itself to the dorso-lateral part of the thalamic mass (fig. 4).

The peripheral collection of cells to which the optic tract passes is the anlage of the dorsal nucleus of the lateral geniculate body. The nucleus increases in width more caudally where the thalamic mass joins the mesencephalon.

Frazer (1940) describes the formation of a distinct chiasmal junction before the end of the second month. He also states that the fibres of the optic tract reach the hinder part of the thalamic formation in the middle of the third month. Gilbert (1935) found that the first nuclear differentiation in the main thalamic body (which she terms the "dorsal thalamus") was the dorsal geniculate nucleus. She recognized it as a poorly defined nuclear mass in a 30 mm. embryo.

Thus at the seventh week of gestation the thalamus has one small differentiated nucleus, the dorsal nucleus of the lateral geniculate body associated with the incoming fibres of the optic tract. The rest of the thalamus is indifferently distinguished into a dorsal part in which cell proliferation is marked, and the ventral part exhibiting less cell formation. The hypothalamic nuclei are forming, and mammillary concentration is progressing. So far only the subthalamic nucleus is recognizable in the subthalamus (fig. 5).

In a specimen of 35 mm. (8½ weeks), the main thalamic mass in its caudal part exhibits further nuclear definition dependent on fibre laminæ. One such band separates very clearly the thalamus from the hypo- and

subthalamic parts, and is the ventral medullary lamina. Continuing from this lamina a band of fibres curves dorsally through the thalamus, and distinguishes a larger ventro-medial region from a narrow lateral region. This band becomes the lateral medullary lamina. The remainder of the thalamus is the dorso-medial portion, but as yet this is not demarcated sharply from the two other parts. Thus the foundations of the future ventral, lateral and dorso-medial thalamic nuclei are established (fig. 6).

The lateral thalamic nucleus forms the dorso-lateral (caudo-lateral) part of the thalamus. An elongated strand of cells at the periphery of this nucleus receives the fibres of the optic tract and is the dorsal geniculate nucleus. If this nucleus be traced in the sections ventrally along the lateral margin of the diencephalon, it will be seen to reach the subthalamus. Here it approximates to a small group of cells which meets the main body of the optic tract. Fibres of the tract pass through the small group and onwards into the whole length of the dorsal geniculate nucleus. The small group of cells is seen to be a subthalamic entity, since it lies ventral to the ventral medullary lamina. It represents the ventral nucleus of the lateral geniculate body (fig. 6).

Gilbert (1935) recognized the ventral geniculate nucleus in a 30 mm. embryo in her series. She found that it originated "at the caudal border of the ventral thalamus." In her terminology the "ventral thalamus" corresponds to the subthalamus or the pars ventralis diencephali.

So far no cell differentiation has occurred in any part of the diencephalon. All the cells are simple neuroblasts with heavily staining nuclei and a minimum of cytoplasm drawn out into one or two processes.

In the fœtus of 40 mm. (9 weeks) the ventral and lateral thalamic nuclei are defined and the latter has enlarged, and in the sections exhibits a dorsal prominence. This becomes the pars posterior of the lateral thalamic nucleus or the pulvinar. The dorsal geniculate nucleus is continuous with the pulvinar and lies ventral to it. The geniculate nucleus is somewhat spindle-shaped and the fibres of the optic tract cover it on its superficial aspect and continue over the surface of the pulvinar. At more caudal levels some of the optic fibres can be traced towards the tectum of the mid-brain. The dorsal geniculate nucleus is clearly removed from the ventral thalamic nucleus, the lateral medullary lamina intervening between them and defining the extent of the ventral thalamic nucleus (fig. 7).

The ventral geniculate nucleus lies ventral to the ventral medullary lamina and thus is regarded as a subthalamic nucleus. Hypothalamic and subthalamic differentiation is progressing especially in the mammillary region, where the mammillo-thalamic tract is well formed.

Nuclear definition in the thalamus is carried a stage further in the 50 mm. fœtus (10 weeks). A curved medial medullary lamina is observed in some specimens, and it demarcates the dorso-medial thalamic nucleus from the ventral and lateral thalamic nuclei. It is identified as the medial lamina by its relation to the fasciculus retroflexus (habenulo-peduncular fasciculus) in the caudal extremity of the thalamus. The lateral thalamic nucleus presents its dorsal geniculate nucleus along its lateral aspect starting just dorsal to the ventral medullary lamina. The dorsal geniculate nucleus continues until it meets the projecting posterior portion of the lateral thalamic nucleus (pulvinar), from which it is faintly demarcated in its caudal part by a slight indentation on the surface. The ventral geniculate nucleus adjoins the dorsal nucleus at the level of the ventral medullary lamina. There is still no differentiation of the thalamic cells (fig. 8).

At subsequent fœtal stages the continuity of the cells of the dorsal projection of the lateral thalamic nucleus (pulvinar) and those of the dorsal geniculate nucleus is clearly seen (figs. 9 to 12). The lateral medullary lamina curves dorso-laterally and distinguishes the ventral and lateral thalamic nuclei, and thus removes the dorsal geniculate nucleus from the ventral thalamic nucleus.

This appearance is not in accord with the belief of Clark (1932*b*), who pronounces the dorsal nucleus of the lateral geniculate body to be a "differentiated lateral portion of the ventral thalamic nucleus." His sketches of the thalamus of a human fœtus of 92 mm. (1932*b*) and of a generalized mammal (1932*a*) do not appear to support his contention. According to these pictures, the dorsal geniculate nucleus is separated from the ventral thalamic nucleus by a very wide medullary lamina. On the other hand his sketches show the dorsal geniculate nucleus and the lateral thalamic nucleus to form a long column around the dorsal and lateral aspects of the thalamus, thus bearing a close relationship to each other topographically. In his sketches Clark indicates a line of demarcation between the lateral thalamic and dorsal geniculate nuclei. This, however, is not visible in the series under consideration, and the two nuclei merge into each other (figs. 9 to 12). The cells even yet do not exhibit much change. Here and there some paler nuclei are observed but they are not confined to any particular thalamic nucleus.

Thus the present research is in agreement with the findings of Gilbert (1935) who states that in the 10·0 cm. fœtus the "internal medullary lamina is distinct and divides the dorsal thalamus into dorsal, ventral and lateral parts. The lateral area includes the dorsal geniculate nucleus and the lateral thalamic nucleus. The latter is still a large undifferentiated mass

of cells, and seems to be directly comparable with the nucleus lateralis and pulvinar of the adult thalamus."

The ventral nucleus of the lateral geniculate body in the 7·0 cm. (12 weeks) fœtus occurs in the form of chains of cells lying among the optic fibres as they diverge to join the dorsal geniculate nucleus. The ventral geniculate nucleus may now be called the nucleus pregeniculatum. No further reference will be made to it (fig. 10).

It will be seen (fig. 1) that the lateral geniculate body projects on the dorso-lateral aspect of the caudal extremity of the thalamus in the 8·2 cm. fœtus (13½ weeks). In the sections it forms a prominent pyriform dorso-lateral mass of cells distinguished dorsally by a small indentation from the posterior part of the lateral thalamic nucleus (i.e. pulvinar), and ventrally by a slight depression from the subthalamus (fig. 11). The medullary laminæ are outstanding, and the ventral thalamic nucleus now presents medial and lateral parts. As already stated, the ventral thalamic nucleus is sharply separated from the dorsal geniculate nucleus by the lateral medullary lamina. Clark (1932*b*) has also drawn attention to the separation of the dorsal geniculate nucleus from the ventral thalamic nucleus by the lateral medullary lamina in primitive mammals. Nevertheless he maintains that the dorsal geniculate nucleus is to be regarded as a differentiated lateral part of the ventral thalamic nucleus.

In all the fœtuses examined between 7·0 cm. and 13·0 cm. (12 to 18 weeks), the continuity of the lateral thalamic nucleus and the dorsal geniculate nucleus is evident. No medullary lamina or fibre band of any description separates the two nuclear structures. It is only by the slight surface indentations that the limits of the dorsal geniculate body can be discerned in sections (fig. 12). Goldby found that in the phalanger the lateral division of the pulvinar could not be distinguished from the medial part of the lateral geniculate body in preparations stained to show the cells. Both consisted of medium and small sized cells of the same type (1940-41).

According to Rioch (1929-30) the lateral geniculate body in the dog is "for the most part demarcated by a fibre capsule, but there is some intermingling of the cells of the two posterior geniculate laminæ with the cells of the lateral portions of the pulvinar and pre-tectal areas." This implies that there is continuity between the lateral geniculate body and the posterior part of the lateral thalamic nucleus (pulvinar).

The cells within the thalamic nuclei are progressing in their development at the 9·0 cm. fœtal stage. There are many small multipolar

neurones to be seen, but still these are not specific to any particular part of the thalamus.

In fœtuses of 13 cm. (18 weeks) the lateral thalamic nucleus has increased in size, and its three divisions "a," "b" and "posterior" can be identified, while its pars geniculatum is the largest of all its nuclei (fig. 12). The continuity of all these parts is evident, and all the constituent cells are alike, being small with very little cytoplasm. In the ventral thalamic nucleus the cells are definitely larger and either spindle-shaped or multipolar. The small cells in the lateral geniculate body are still evenly dispersed and closely crowded among the optic fibres (fig. 13), whereas in the ventral thalamic nucleus they are less congested.

In fœtuses of about 17 cm. length (22 weeks) a modification of the pyriform shape of the lateral geniculate body is noticeable. The dorso-medial wider aspect possesses a V-shaped indentation from which fibres issue (fig. 14). No further structural change is observed until the sixth month of gestation (19 to 20 cm.). At this stage lamination of the lateral geniculate body is observed. At first only the cephalic two-thirds are affected, the caudal third still retaining its original even dispersal of cells. From the outset the laminæ show a U-or V-shape. They consist of small differentiated neurones, and are incompletely separated by narrow fibrillary zones. The disposition of the laminæ shows a high degree of similarity in all cases. When examined in sections cut in the plane of the optic tract, the convex aspect of the largest and outermost curved lamina is presented ventro-laterally and receives the fibres of the optic tract which diverge to enter the convexity of this lamina. Some optic fibres encircle the lateral geniculate body. Next follow three smaller laminæ curved in conformity with the first, and likewise presenting their convexity towards the entering optic tract. In these four laminæ the lateral limb of the U is longer than the medial one, and hence the open part of the laminæ looks dorso-medially. The lamination is completed by two smaller and narrower laminæ lying in the open mouth of the other U-shaped ones. These smaller laminæ are wide open V-shaped. Thus the concavity of the laminæ is always looking in a dorso-medial direction, away from the entrance of the optic tract. The concavity corresponds in position to the indentation seen in fig. 14. The optic tract fibres enter the convexity of the laminæ, while from the laminar cells new fibres arise which emerge on the concave aspects of the laminæ and form the fibrillary strata which alternate with the laminæ (fig. 15). These strata are therefore composed of the optic radiations (Glees and Clark, 1940-41). The outermost and largest lamina is itself indented at its dorso-lateral extremity, and from this

indentation fibres emerge which run dorsally over and through the pulvinar towards the superior quadrigeminal body. In a recent paper Clark (1940-41) states that the laminar differentiation in the human lateral geniculate body occurs sometime between the fourth and sixth fœtal months.

Throughout the remainder of fœtal life and in infancy and childhood the pattern of the laminæ of the lateral geniculate body is the same. Minor differences depend on the passage of the optic tract fibres through the outer laminæ in order to reach the inner ones. The diversity in the laminar pattern may depend to some extent on the junction of separated portions of one lamina with those of another. The laminæ at first consist entirely of small neurons, but in fœtuses of eight months' gestation the cells of the two small innermost laminæ are larger than those in the four outer laminæ (fig. 16).

The disposition of the geniculate laminæ as seen in the present investigation is totally different from that described and figured by Woollard and Beattie (1926-27), Clark (1932*b*), and others. In his earlier papers Clark (1932*b*), stated that the lateral geniculate body consisted of six laminæ, when seen in coronal or transverse sections, four larger U-shaped ones lying dorsally and capping two smaller ventral ones. The larger laminæ he found to consist of smaller cells and the smaller laminæ of larger cells. The open ventral aspect of the laminæ he called the hilum. He described and figured optic tract fibres and blood vessels entering this hilum and spreading out dorsally in radial fashion to reach the concave ventral aspects of the laminæ. Certain other workers adopted Clark's views, amongst them Abbie (1934), Mackenzie (1934), and Clark and Penman (1934). Abbie (1934) described and figured blood vessels entering the ventral hilum with the optic tract and radiating dorsally along with the optic tract fibres. Clark and Penman (1934) mention a process of "eversion" of the laminæ during development which produces a medio-ventral hilum into which the optic tract enters, and a dorso-lateral convexity from which the optic radiations emerge. It has been shown in the present paper that in the human fœtus the organization of the laminæ is opposite to that described by Clark, the optic tract fibres entering their convexity and the optic radiations emerging from their concavity. There is certainly no ontogenetic "eversion" in the human lateral geniculate body.

In his morphological study of the lateral geniculate body, Clark (1932*b*) surveyed this body in a graded series of members of the Lemuroidea. He states that in the mouse lemur the laminæ show a curvature conforming

to the convex lateral surface of the thalamus. In the dwarf lemur a tendency towards the incurving of the laminæ is more apparent, and a medial concavity is better defined from which the optic radiations emerge. He called this arrangement of the geniculate laminæ an "inversion." On the other hand Clark asserted that in the Anthropoidea and all monkeys the laminæ become completely "everted," and as a result "a definite hilum is formed, directed ventro-medially, and it is from this direction that optic tract fibres, as well as branches of the posterior cerebral artery, enter the lateral geniculate body." As regards man and the African anthropoid apes, Clark found the eversion was less complete "since the original dorsal and ventral margins of the dorsal nucleus of the lateral geniculate body have not become approximated to complete the formation of a hilum."

In his work on the diencephalon of the cat, Thuma (1928) described and illustrated the lamination of the lateral geniculate body and made a reconstruction. He found the laminæ to be S shaped, and in his figure showed that the optic fibres entered the large lateral convexity of the laminæ. The appearance in the human lateral geniculate body has been seen in the present investigation to accord with that described by Thuma.

Since the commencement of the present investigation of the development of the human diencephalon and mid-brain nuclei, Glees and Clark (1940-41) have examined the mode of termination of optic fibres in the lateral geniculate body of the rhesus monkey. In their paper these authors state that "it was found most useful in order to follow the course of the optic fibres to cut the lateral geniculate body in the *vertical* plane along the axis of the optic tract." They submit a diagram and add, "from the diagram it becomes clear that the bulk of the optic fibres do not enter the lateral geniculate body from the hilum; on the contrary they penetrate the curved laminæ on their convex aspect, many of them passing through the greater part of the geniculate body in order to reach the cells at the hilum." In the legend accompanying the diagram these authors state "it will be noted that in this plane the hilum of the lateral geniculate body is at its caudal end while the optic tract fibres penetrate it from its convex aspect."

In spite of the earlier emphasis by one of these authors (Clark, 1932*b*) on the entrance of the optic tract fibres into the concave ventral hilum of the laminated lateral geniculate body, no mention in the joint paper is made of these previous descriptions, and no explanation is given why a completely contradictory view has been put forward. In a later paper Clark (1940-41) describes the lateral geniculate body of the macaque monkey

as it appears in a reconstruction from serial sections cut transversely to the optic tract. He makes little reference to the manner in which the optic tract fibres enter the convex aspect of the laminæ of this body.

Owing to this modification of views in the above-mentioned work, it was decided to review the whole question of the lamination in the lateral geniculate body in the light of information obtained during the investigation of the development of the human diencephalon. For this purpose serial sections were cut of the lateral geniculate body *in situ* in human, old world monkey and new world monkey brains. The sections were made (*a*) in the plane of the optic tract, (*b*) in the coronal plane of the entire brain, (*c*) in the sagittal plane of the entire brain.

The dorsal nucleus of the lateral geniculate body in the new world monkey (Feline douroucolli) contains only four laminæ, two larger U-shaped ones embracing two smaller V-shaped ones. The larger outer laminæ consist of small cells, and their convexity is directed towards and receives the optic tract. The smaller inner laminæ are made up of large cells and they likewise receive optic tract fibres on their convex aspects. The concavities of the four laminæ face dorso-medially. As the optic tract approaches the geniculate body its fibres fan out in three ways: (1) the majority spread over the entire outermost lamina and penetrate it at all points to reach the inner laminæ: (2) the peripheral fibres envelop the geniculate body: (3) finally, a bundle of fibres passes directly through all four laminæ in the centre of the body (fig. 17) and joins those fibres which have encircled the geniculate body and those which have emerged from the cells in the laminæ. It appears that the penetrating bundle interrupts the continuity of each lamina, but it could not be ascertained in this series if any of the fibres terminated in the laminæ. Followed beyond the lateral geniculate body this bundle can be traced towards the pulvinar and tectum.

The lateral geniculate body of the old world monkey (Barbary ape) was found to be similar to that of the human subject. Six laminæ are present, four larger outer ones and two smaller inner ones. In the larger laminæ small cells occur, while larger cells are present in the two smaller laminæ.

The blood supply was examined in the sections of the lateral geniculate body. It was seen that while small blood vessels entered the body along with the optic tract, larger ones were concentrated in the dorso-medial aspect especially in relation to the innermost large-celled laminæ (fig. 18). These vessels arise from the posterior choroidal artery and they gain this position by penetrating the dorso-medial indentation (see below) of the

lateral geniculate body, just lateral to the medial geniculate body. Abbie (1934) found that the posterior cerebral artery provides branches which are distributed in the postero-medial part of the lateral geniculate body, while the anterior choroidal artery supplies the lateral aspect of the body. Free anastomoses occur between the two systems.

If the external features of the lateral geniculate body and optic tract are examined, it will be seen that the tract enters the rostro-lateral aspect of the body. On the caudo-medial aspect a well-defined V-shaped indentation is apparent which faces and comes into contact with the lateral aspect of the pulvinar. This indentation corresponds to the open concavities of the laminæ, and here enter branches of the posterior choroidal artery.

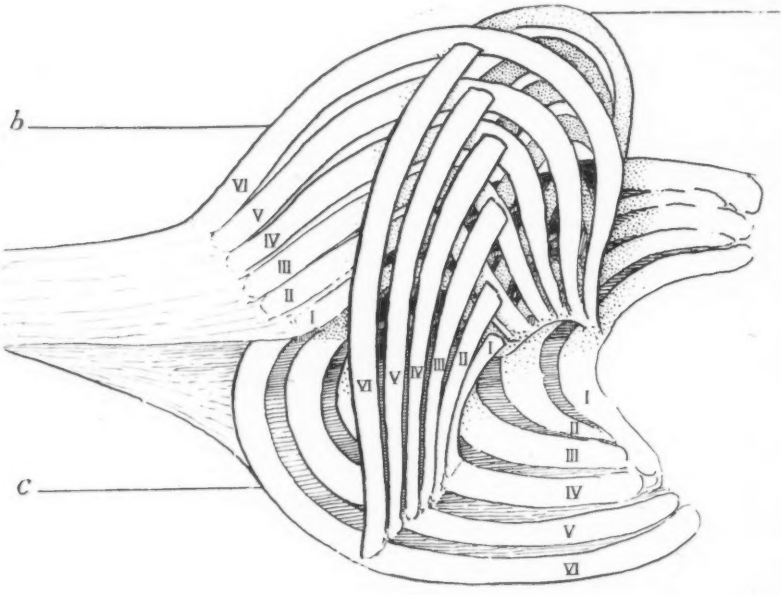

FIG. 19

FIG. 19 represents diagrammatically the appearances in the middle of three adult human geniculate bodies cut *in situ* respectively: (1) in the plane of the optic tract (fig 18); (2) in the coronal (frontal) plane; and (3) in the parasagittal plane (fig. 20). By reconstruction from the serial sections cut in these planes it is seen that the lateral geniculate body consists of four large and two smaller laminæ disposed one inside each other.

The innermost smaller laminæ are somewhat bell-tent shaped, their wide-open bases facing dorso-medially and lying caudally. The four larger laminæ are cup-shaped, but the medial walls are shorter than the lateral, and likewise the open aspects are directed dorso-medially. The outermost largest lamina extends most rostrally and caudally, and each of the other five reaches rostrally and caudally to a lesser extent. The innermost smallest lamina forms a core around which the others are disposed. Clark (1932*b*) calls this core the hilum. The convex aspect of each lamina receives the optic tract fibres which then spread over the convexity in a caudal and dorsal direction. Sections in the sagittal plane show that, on joining the lateral geniculate body, the optic tract sends its most ventral fibres to the core or innermost lamina, while its most dorsal fibres curve dorsally to join the outermost lamina. Between these two, fibres are distributed to the other laminæ in the organized manner (fig. 20).

Thus each lamina with its appropriate band of fibres from the optic tract presents a spoon-shaped appearance, and the six fit one inside the other. Clark (1940-41) states that the crossed and uncrossed fibres of the optic tract do not become segregated until after they have penetrated into the lateral geniculate body.

From the appearances in this representative series of the lateral geniculate body, it is apparent that the term "eversion" as applied by Clark to the orientation of the laminæ in the anthropoid and human lateral geniculate body is meaningless (1932*b*). According to his own observations on the lemurs, and those of the present research, the lamination is the same in all cases. The laminæ are arched and fit one inside the other. The convex aspects of the arches curve laterally and dorsally in relation to the entire brain, and conform to the convex superficial surface of the lateral geniculate body. The difference between the laminar arrangement in the lemurs and in the anthropoids (as seen in sections) is not due to any alteration in the direction of the laminæ, but depends on a greater degree of curvature. In other words the laminæ in the lemurs form small arcs of relatively large circles, while those in the anthropoids and man form large arcs of relatively small circles.

The entrance of the optic fibres into the arched or convex surfaces of the geniculate laminæ is not unique. A comparable arrangement is found in the bulbar olive and the cerebellar dentate nucleus, both of which receive fibres on their convexity and emit fibres from their concavity.

Clark's figures (1932*b*) of the lateral geniculate bodies of a woman whose left eye had been removed for glaucoma are not easy to orientate. The geniculate bodies in this case were excised and sections were made

in the coronal plane. Leaving out of account his misinterpretation of the relation of the optic tract to the laminæ, as the figures are presented it is not conceivable that in the right and left lateral geniculate bodies the swing of the curved laminæ should be over to the right on both sides. Nor is it likely that the hilum, which he wrongly supposed received the optic tract fibres, should be on the left side in both right and left geniculate bodies.

It has been shown that the human lateral geniculate body is closely identified embryologically and topographically with the lateral thalamic nucleus. Optic fibres reach the geniculate body and some proceed over or through the pulvinar (posterior part of the lateral thalamic nucleus) to gain the superior colliculus. The optic radiations are carried to the area striata of the cerebral cortex. In lower vertebrates the optic tectum is the main termination for fibres of the optic tract. Is it conceivable that, with the phylogenetic development of the telencephalon, the part of the optic tectum which subserves visual functions (not that part concerned with reflexes) is moved in a cephalic direction away from the tectum and orientated as a part of the thalamus? In other words, is the lateral geniculate body originally a mesencephalic structure which during phylogenetic development is shifted forwards until ultimately it becomes incorporated within and developed along with the lateral thalamic nucleus?

Clark (1942) states that primarily the lateral geniculate body appears to be functionally subordinate to the tectum as a visual centre.

The author wishes to thank Professor F. Wood Jones for his welcome and generous interest, advice and criticism during this investigation and preparation of the paper. Thanks are also due to Mr. H. Gooding who prepared the photographs with his usual skill and care.

Summary and Conclusions.

(1) The human lateral geniculate body (pars dorsalis) differentiates in the lateral thalamic nucleus, while the pars ventralis is subthalamic in origin.

(2) The pars dorsalis is recognizable in the embryo of 22 mm., and arises as the optic tract reaches this region of the thalamus. It is the first nuclear mass to be distinguished in the thalamic portion of the diencephalon. The pars ventralis is first seen at 35 mm.

(3) No lamination is evident until the 6th month of gestation.

(4) From the outset there are six U- and V-shaped laminæ which present their closed convex aspects ventro-laterally and receive the fibres of the

optic tract. From the dorso-medially directed concavities issue the optic radiations.

(5) The four larger outermost laminæ consist of small cells, while the two smaller innermost laminæ contain large cells.

(6) There is no such thing as "eversion" of the laminæ in the human or monkey lateral geniculate body as originally described and figured by Clark and his associates. The laminæ are curved to a lesser degree in the lemurs and to a greater degree in man and the monkeys, but all are curved in the same direction, the convexity always receiving the optic tract.

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KEY TO ILLUSTRATIONS.

1.—Cerebral vesicle.
2.—Pars dorsalis thalami.
3.—Pars ventralis thalami.
4.—Hypothalamic sulcus.
5.—Subthalamic nucleus.
6.—Hypothalamus.
7.—Spread of cells towards fibres of optic tract.
8.—Fibres of optic tract.
9.—Cell free zone between pars dorsalis thalami and pars ventralis thalami.
10. Dorsal nucleus of lateral geniculate body.
11.—Mammillary concentration.
12.—Ventral thalamic nucleus.
13.—Ventral medullary lamina.
14.—Mammillo-thalamic tract.
15.—Wall of cerebral hemisphere.
16.—Lateral thalamic nucleus.
17.—Lateral medullary lamina.
18.—Ventral nucleus of lateral geniculate body.
19.—Crus.
20.—Pulvinar.
21.—Dorso-median thalamic nucleus.
22.—Medial medullary lamina.
23.—Internal capsule.
24.—Nucleus entopeduncularis.
25.—Substantia nigra.
26.—Medial geniculate body.
27.—Fibres emerging from indentation of lateral geniculate body.

PLATE XII.

FIG. 1 9.2 cm. 8.2 cm. 7.0 cm. 6.0 cm.

FIG. 2

FIG. 3

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PLATE XIII.

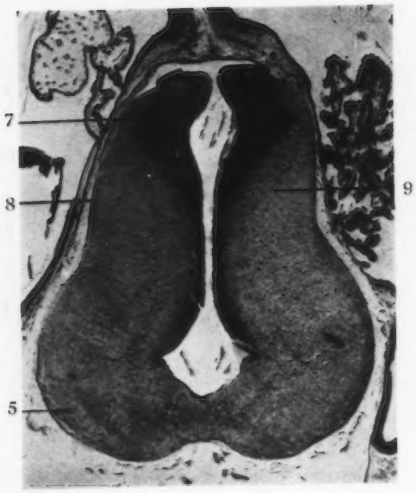

FIG. 4

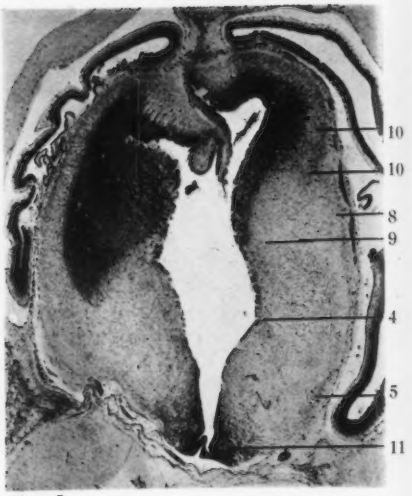

FIG. 5

FIG. 6

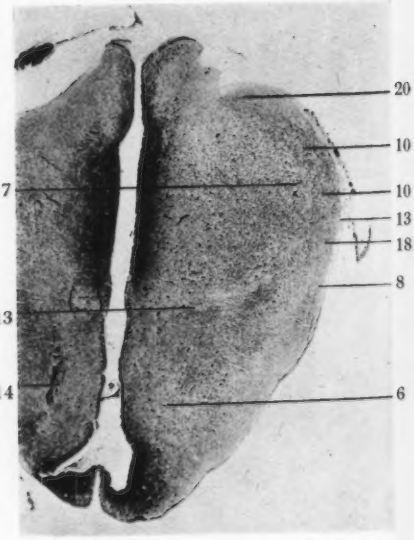

FIG. 7

To illustrate article by E. R. A. Cooper

PLATE XIV.

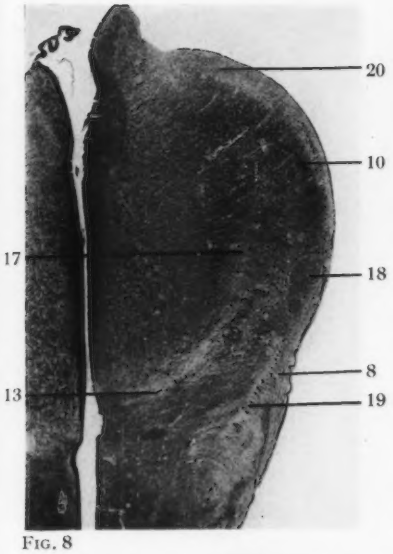

FIG. 8

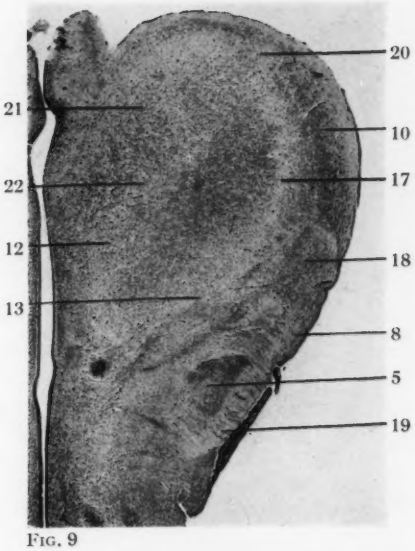

FIG. 9

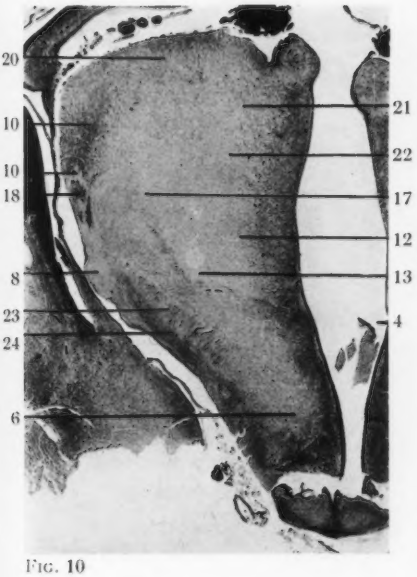

FIG. 10

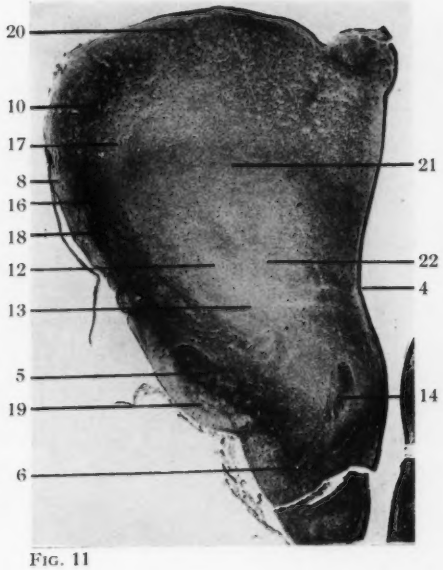

FIG. 11

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PLATE XV.

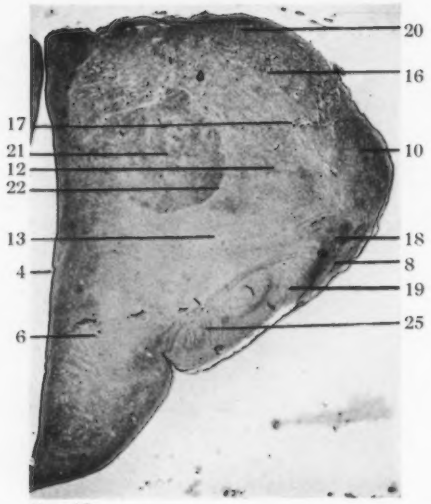

FIG. 12

FIG. 13

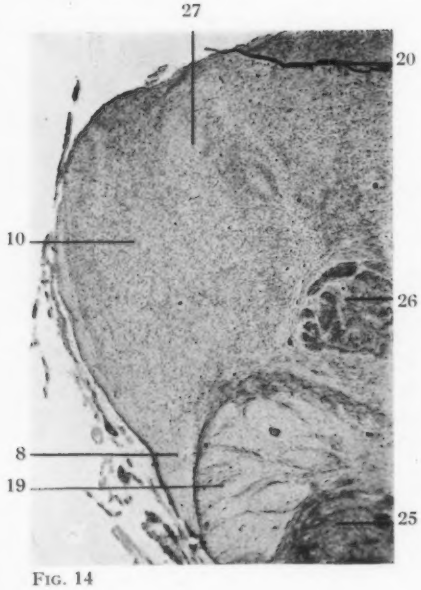

FIG. 14

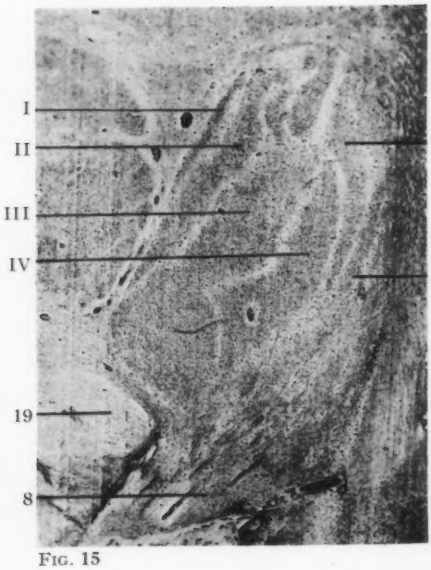

FIG. 15

To illustrate article by E. R. A. Cooper

PLATE XVI.

FIG. 16

FIG. 17

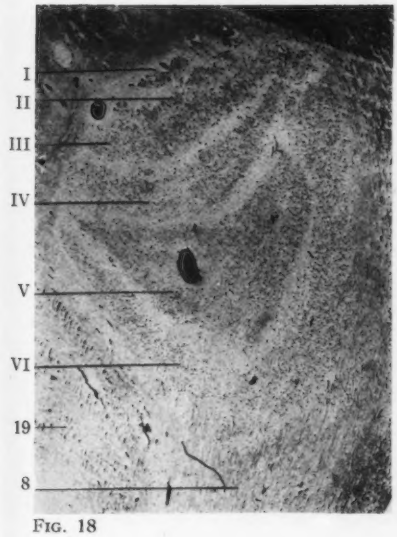

FIG. 18

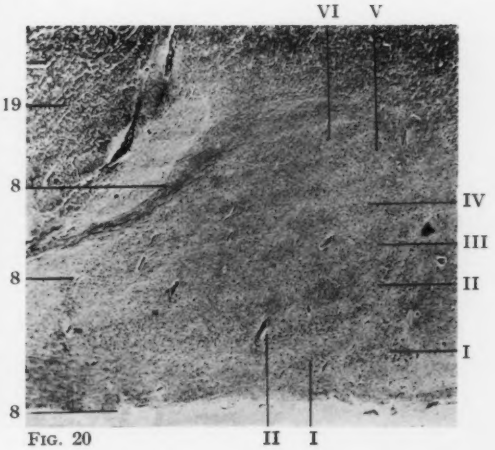

FIG. 20

To illustrate article by E. R. A. Cooper

LEGENDS.

FIG. 1.—Brain stems of fœtuses of 9·2, 8·2, 7·0 and 6·0 cm. length (*a*)—lateral geniculate body; (*b*)—medial geniculate body.

FIG. 2.—Diencephalon with optic outgrowths. 8·5 mm. embryo. × 35.

FIG. 3.—Diencephalon. 16 mm. embryo. × 40.

FIG. 4.—Caudal region of diencephalon. 22 mm. embryo. × 20.

FIG. 5.—Diencephalon. 24 mm. embryo. × 25.

FIG. 6.—Diencephalon. 35 mm. embryo. × 20.

FIG. 7.—Diencephalon. 4·0 cm. fœtus × 18.

FIG. 8.—Diencephalon. 5·0 cm. fœtus. × 18.

FIG. 9.—Diencephalon. 6·3 cm. fœtus. × 15.

FIG. 10.—Diencephalon. 7·0 cm. fœtus. × 13.

FIG. 11.—Diencephalon. 8·2 cm. fœtus. × 16.

FIG. 12.—Diencephalon. 13 cm. fœtus. × 10.

FIG. 13.—Fibres of optic tract dispersing among the cells of the lateral geniculate body. No lamination yet. × 200. (Silver impregnation.)

FIG. 14.—Lateral geniculate body. 17·5 cm. fœtus. × 17.

FIG. 15.—Lateral geniculate body. 6½ months' fœtus. × 20. Laminæ I to VI.

FIG. 16.—Lateral geniculate body. Full term fœtus. × 12. Laminæ I to VI.

FIG. 17.—Lateral geniculate body. New world monkey. × 20. Laminæ I to IV.

FIG. 18.—Lateral geniculate body. Adult. Sectioned in the plane of the optic tract. × 12. Laminæ I to VI. Note blood vessels near lamina I.

FIG. 19.—Representation of the lateral geniculate body sectioned in the coronal plane, parasagittal plane and plane of the optic tract. Laminæ I to VI.

FIG. 20.—Parasagittal section of lateral geniculate body. Adult. Laminæ I to VI. × 10.

Notices of Recent Publications.

Recent Progress in Psychiatry. Being the Journal of Mental Science for January, 1944, vol. xc, No. 378.

Under this title a number of reviews on subjects of interest to psychiatrists have been collected in a volume of some 500 pages. The reader will find, however, a note of paradox in the title, for a number of the articles are reviews of the anatomy and physiology of the brain, of pathology and of neurosurgery. Doubtless the knowledge so summarized is of value to the psychiatrist, now that he has come to see the relevance of neuro-anatomy and physiology in treatment, but it represents progress in these subjects by workers in the relevant fields and cannot, save by that metaphor in which medical psychology is so rich, be regarded as progress in psychiatry. Such are the chapters by Le Gros Clarke on the anatomy of the nervous system, by Richter on the biochemistry of the nervous system, by Grey Walter on electro-encephalography, by Meyer on neuropathology and by Nicol on the treatment of neurosyphilis. In these, as in the more appropriately so-called psychiatric chapters, the reader will find a valuable collection of critical essays which will be of use to those who have no time to consult original sources, to follow research for themselves, or to make their own judgments upon recent work in all the fields touched upon.

In the introductory chapter by the editor there is a note of naïve hopefulness for the future of psychiatry that gives no hint of the intellectual antinomy that now appears in this complex and increasingly ill-defined field of thought, where causation and mode of disorder are still by hypothesis purely psychological, but treatment increasingly physical.

Neurology of the Eye, Ear, Nose, and Throat. By E. A. Spiegel and I. Sommer. Grune and Stratton, New York. Pp. 690. Price 7 dollars and 50 cents.

This book, based upon postgraduate lectures given by the authors, first in Vienna and later in America, sets out to outline the anatomy, physiology, pathology and nosology of the nervous mechanisms of the eye, ear, nose and throat, and to describe the methods of functional testing of these organs, the diagnosis and treatment of their neurological disorders.

Yet it is probably not for pathology, diagnosis or treatment that the reader will value this work of reference, for on all these subjects the matter is incomplete, and given too much in the manner of a short encyclopædia than in the true manner of the clinical textbook of the first rank.

Its best section is probably that dealing with the physiology of the labyrinth, and this because Magnus and his pupils expounded this so lucidly and with so striking a harmony of observation and synthesis that it would be difficult for any careful précis writer not to make it clear. Where, however, previous observers and writers have not shown these gifts consistently, as, for example, in the matter of the analysis of cerebellar disorders and in that of the functions and disorders of the cerebral cortex, the accounts given here are heavy, burdened with inadequately correlated facts, and form an ill-digested and sometimes tedious exposition, and one showing no real critical faculty.

Nevertheless, the advanced reader will find it a valuable work of reference and a quarry wherein he may profitably dig for factual information on the range of subjects with which it deals.

Illustrations of Regional Anatomy. By E. B. JAMIESON. Sixth edition. Livingstone, Edinburgh. Price 75s.

All students will give a warm welcome to the appearance, bound within a single cover, of the seven sections of Jamieson's now familiar illustrations. That it should have been possible to produce during wartime a volume of over three hundred pages of coloured anatomical drawings is a striking tribute to all concerned, anatomist, artist, printers and publisher.

Of special interest to the neurologist will be the illustrations of the central and peripheral nervous system, and of the anatomical relationships of the latter. They form an ample and well-chosen series, and a great many of the other figures, as for example the view of the root of the neck on p. 107, will prove useful to the working neurologist, whose library might well contain this volume.

Psychological Medicine. A Short Introduction to Psychiatry. By DESMOND CURRAN and ERIC GUTTMANN. Second edition. 1945. Livingstone, Edinburgh. Price 10s. 6d.

The first edition of this concise and lucid manual for practitioners and students was reviewed in this journal in 1943 (vol. 66). In its revised and slightly enlarged second edition it should continue to command a wide popularity. It is perhaps ungracious to make a minor criticism of so excellent a publication, but one wonders what useful purpose is supposed to be served by a photograph (fig. 4) of a box with knobs and dials on its surface, behind which, the reader is assured, lies the apparatus for the induction of electrical convulsions. This object becomes increasingly familiar in psychiatric works, and like the caskets in the Merchant of Venice it completely conceals its secrets. It remains but to inscribe on it "who chooseth me shall get as much as he deserves."

The Shaping of Psychiatry by War. By J. R. REES. Chapman and Hall, London. 1945. Price 10s. 6d.

In these pages the reader will not seek for a more profound thought, or for that documented assessment of the subject dealt with, than is reasonable in a series of memorial lectures. He will find in them a very readable account of what has been attempted and done by the author and his colleagues in the British Army in the matter of intelligence grading and in selection of men for the various ranks and tasks that a modern army at war demand.

The management of psychological disorders in combatants, and the future of psychiatry, its relations to government and to social and to other aspects of medicine are also discussed.

The texture of the author's thought is revealingly displayed throughout his pages. For example, he says that "most doctors are individualists and are proud of it." Yet, lest we become too proud, he hastens to assure us that "the four professions most liable to be chosen by those with marked feelings of inferiority are the law, the Church, teaching and medicine, all professions in which you talk down to people and in which they don't answer back." This is a quaint conception of medicine, and having thus airily swept all the learned professions off the board, it is likely that Dr. Rees does not expect his readers to answer him back.

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